JUN 5 1947
MEDICAL LIBRARY

ACTA PÆDIATRICA

EDITORES:

A. LICHTENSTEIN, STOCKHOLM, A. WALLGREN, STOCKHOLM

REDACTORES:

IN DANIA: BENT ANDERSEN, AARHUS, OLUF ANDERSEN, KØBENHAVN, C. E. BLOCH, KØBENHAVN, P. PLUM, KØBENHAVN. *IN FENNIA:* P. HEINIÖ, HELSINGFORS, V. RANTASALO, HELSINGFORS, C.-E. RÄIHÄ, HELSINGFORS, T. SALMI, ÅBO, ARVO YLPPÖ, HELSINGFORS. *IN HOLLANDIA:* E. GORTER, LEIDEN, CORNELIA DE LANGE, AMSTERDAM, J. VAN LOOKEREN CAMPAGNE, GRONINGEN. *IN NORVEGIA:* TH. FRÖLICH, OSLO, LEIF SALOMONSEN, OSLO, L. STOLTENBERG, OSLO, A. SUNDAL, OSLO, KIRSTEN UTHEIM-TOVERUD, OSLO. *IN SUECIA:* C. GYLLENSWÄRD, UPPSALA, N. MALMBERG, STOCKHOLM, STURE SIWE, LUND, WILHELM WERN STEDT, STOCKHOLM, Y. ÅKERRÉN, GÖTEBORG.

REDIGENDA CURAVIT
A. LICHTENSTEIN
KRONPRINSESSAN LOVISAS BARNSJUKHUS,
STOCKHOLM

Vol. XXXIII. Supplementum 1

1946

Almqvist & Wiksells Boktryckeri Aktiebolag
UPPSALA 1946

ACTA PÆDIATRICA

PROFESSOR A. LICHTENSTEIN
KRONPRINSESSAN LOVISAS BARNSJUKHUS,
30 POLHEMSGATAN, STOCKHOLM

The 'ACTA PÆDIATRICA' contain articles relating to pediatrics. These articles are published in English, French or German, according to the wishes of the author. Each number consists of about 6 printed sheets, 4 numbers forming a volume. The numbers will be issued as soon as the articles sent in can be printed. The 'Acta' is open to articles from foreign authors in all countries, if sufficient space can be found for them. Manuscripts are to be sent direct to the Editor, to whom also enquiries about the exchanging of papers are to be directed. The subscription should be forwarded to the Editor. Each volume costs 25 Swedish crowns or 25 shillings or 5 dollars.

ACTA PÆDIATRICA enthalten Arbeiten aus dem Gebiete der Kinderheilkunde. Die Arbeiten werden, je nach eigener Wahl des Verfassers, in deutscher, französischer oder englischer Sprache veröffentlicht. Jedes Heft enthält cirka 6 Druckbogen; 4 Hefte bilden einen Band. Die Hefte erscheinen, je nachdem die in dieselben aufzunehmenden Aufsätze druckfertig vorliegen. Die Acta nehmen nach Möglichkeit auch Arbeiten ausländischer Verfasser aller Nationen auf. Manuskripte nimmt der Herausgeber entgegen, desgleichen Wünsche betreffs Austausch von Zeitschriften. Abonnementanmeldung bei dem Herausgeber. Preis pro Band 25 schwedische Kronen.

Les ACTA PÆDIATRICA contiennent des ouvrages du domaine de la pédiatrie. Les études sont publiées en français, anglais ou allemand au choix de l'auteur. Chaque fascicule contient env. 6 feuilles in -8°; 4 fascicules forment un volume. Les fascicules paraissent au fur et à mesure que les articles y destinés sont imprimés. Les Acta reproduisent, dans la mesure du possible, les articles d'auteurs étrangers de tous les pays. Les manuscrits doivent être expédiés à l'éditeur, à qui les demandes relativement à l'échange de journaux devront également être adressées. Abonnement chez l'éditeur. Prix par volume Cr. Suéd. 25.

ACTA PÆDIATRICA

ACTA PÆDIATRICA

Because of an oversight the numbers and the titles of the supplements in Vol. XXXIII (1946) were omitted. They are as follows:

I. Olle Elgenmark: The Normal Development of the Ossific Centres during Infancy and Childhood. A clinical, roentgenologic, and statistical study. 79 pp.

II. Gert v. Sydow: A Study of the Development of Rickets in Premature Infants. 122 pp.

III. Kurt Kaijser: Gastric Juice Examinations in Children. A clinical-physiological study. 57 pp.

IV. Siv Gunnarson: Some Types of Nervous Disorders in Children and their Prognosis. An investigation based on the study of hospital material, followed up by subsequent examination. 76 pp.

V. Edith Otterström: Delinquency and Children from Bad Homes. A study of their prognosis from a social point of view. 326 pp.

VI. Lars-Erik Carlgren; Gallop Rhythm in Children. Studied by means of calibrated phonocardiography. An experimental and clinical investigation. 141 pp.

VII. Erik Jacobsson: Rheumatic Fever with Chorea Minor. A clinical study with special reference to the prognosis. 222 pp.

From The Samariten Children's Hospital, Stockholm
Head: Docent Nils Malmberg, M. D.

From The State Institute of Human Genetics and Race Biology, Uppsala
Head: Professor Gunnar Dahlberg, M. D., LL. D.

THE NORMAL DEVELOPMENT OF THE OSSIFIC CENTRES DURING INFANCY AND CHILDHOOD

A CLINICAL, ROENTGENOLOGIC, AND STATISTICAL STUDY

BY

OLLE ELGENMARK

ACTA PÆDIATRICA, VOL. XXXIII, SUPPLEMENTUM I

Stockholm 1946

UPPSALA 1946
ALMQVIST & WIKSELLS BOKTRYCKERI AB
46121

To my Wife

Medical
direct

CONTENTS.

		Page
Preface		7
Chapter 1.	Survey of Literature	9
» 2.	Material and Methods	22
» 3.	Relation between Ossification and Age	31
» 4.	Comparison between Skeletal Development in the two Halves of the Body	53
» 5.	Correlation Calculations	56
» 6.	Normal Tables for the Development of the Ossific Centres	61
Summary		76
References		78

PREFACE.

The development of the ossific centres in early childhood has not previously been studied in the northern countries. Standard tables compiled abroad have therefore been used for the estimation of skeletal development in this country. It is, however, conceivable that because of race and environmental differences the figures relative to skeletal development applying to one people may not be equally applicable to the people of another nation. It has, in fact, been found that in Sweden satisfactory results are not obtained with these tables, which in many cases are not based on uniform material or on adequate statistical calculations. A study of the literature reveals that our knowledge concerning the development of the ossific centres is in many regards defective.

In the present work the author has therefore endeavoured to throw light on a number of problems connected with the skeletal development, and to work out a set of normal figures for this country on the basis of the results obtained.

The clinical and roentgenographic examinations were carried out at the Samariten Children's Hospital, Stockholm, and I should like first of all to express my sincere thanks to the head physician of the hospital, Docent NILS MALMBERG, for suggesting to me the lines on which an investigation of this kind might be conducted, as well as for all the advice and encouragement which he extended to me during the entire course of the work. My gratitude is also due to Docent FOLKE KNUTSSON, who advised me as to a suitable technique for the roentgen examinations and throughout my studies was always ready to assist me in the interpreting of the roentgenograms. The interest shown by Professor ÅKE ÅKERLUND in my investigation is also greatly appreciated. I should also like

to thank Docent SVEN-ROLAND KJELLBERG for the valuable help he gave me in connection with the roentgen examinations.

The statistical calculations were carried out at the State Institute for Human Genetics and Race Biology at Uppsala and I take this opportunity to proffer my grateful thanks to the head of the Institute, Professor GUNNAR DAHLBERG, for giving me working place at the institute and placing every assistance at my disposal. Above all I must thank him for freely giving me advice and help.

To my colleagues at the Samariten Children's Hospital, and members of the hospital staff who aided me in various ways with my investigation, I am grateful for friendly cooperation. I also owe a debt of gratitude to the staff of the Institute for Human Genetics, Uppsala.

Mrs. GRACE LUNDBERG has bestowed careful work on the translation and the proof-reading, for which I desire to express my thanks.

The investigation was carried out with the aid of grants from »Stiftelsen Therése och Johan Anderssons Minne».

Stockholm, April 1946.

Olle Elgenmark.

CHAPTER 1.

Survey of Literature.

As soon as radiography began to assume practical importance as one of our most valuable diagnostic aids its value for the purpose of increasing our knowledge regarding the growth and development of the skeleton was quickly realized. Previously, it had only been possible, in anatomic studies, to establish the presence of small ossified regions which gradually developed. It was only with the introduction of roentgenography that it became possible to obtain more exact information regarding the development of the skeleton from the first appearance of ossific centres to the synostosis between epiphysis and diaphysis which signifies the cessation of the growth.

The first reports on investigations on ossification from ossific centres were published within a few years of the discovery of the roentgen rays. Mention may be made here of works by BEHRENDSEN (1897), and VON RANKE (1898), and several publications by BADE, HAHN, and VON WYSS (1899), and during the first part of the twentieth century by WILMS (1902), HASSELWANDER (1903) and HOLMGREN (1906).

In all of these investigations only one special ossification area was selected for study, usually the hand or foot, and it was believed that it was possible, on the basis of the results obtained, to draw definite conclusions regarding the development of the skeleton as a whole.

The material for study was small and incomplete, the presence of disease was not taken into consideration, and no attention was paid to the factors which, according to more recent researches,

have proved to be of vital significance for a judgment on the growth of the skeleton.

In the years that followed, it was found that the connection between the development of the ossific centres and the year of life corresponding to it was not always what had been expected, and closer investigation proved that the causes of these discrepancies were to be found in variations in such factors as height, weight, heredity, environment, and diseases.

Because of the essential nature of a knowledge of these factors for the study of ossification I have treated each of these factors separately in this survey.

In all investigations hitherto made, the ossification has been put in relation to *age* without any discussion as to the suitability of this method. STETTNER (1921) writes that he found in his studies of the bones of the hand that no new ossific centre appears in the wrist between the ages of 1 and 2 $^1/_2$ years, and that during this period one cannot reach any conclusions regarding the stage of skeletal development in relation to age from a study of this part alone, as is often done. His material of 203 boys and 212 girls, almost all of whom were around the age of 2, is so large that his observations must be taken as significant, despite the fact that he has no statistical background for his statement.

STUART and VICKERS (1936) express a doubt, in connection with their investigation on the skeleton of the hand, that any conception of the skeletal development can be gained from the age alone, or vice versa. In their material, consisting of children up to the age of 5, they observed long periods of stagnation in the ossification process, but no further details are given regarding the length of these periods, nor is there any amplification of the material with a view to explaining this observation.

As early as 1899, VON WYSS thought he could observe a connection between *body height* and ossification in his studies of the development of ossific centres in cretins. His investigation was confined to a few cases only, and in these he found that the skeleton was the same, with respect to the development, as in healthy normal persons of the corresponding height.

In the same year, HAHN published a report on a roentgeno-

graphic investigation carried out for the purpose of studying the epiphyseal centre at the proximal end of the tibia in newborn, well-nourished infants. He was able to distinguish the centre in roentgen films if the length at birth was not below 49 cm, and this he regarded as indication of the fact that there is a close connection between the height and the date of appearance of the ossific centre. No mention is made of the number of cases in the different height groups, and this statement in his publication thus possesses little value. In 1910, HOLMGREN described an investigation on ossification in the bones of the hand in exophthalmic goiter. In a material of 113 roentgenograms from persons ranging between the ages of 10 and 20 years he found such a consistent correspondence between increase in height and ossification that it could not be put down to chance alone, and he writes: »in persons of the same age the ossification shows just as much of an advance as the individual is taller», and regarding persons of different ages he adds: »older individuals of shorter stature often display the same development as younger ones who are taller».

Holmgren had observed in connection with his investigation that increased glandular function caused a more rapid increase in height, and it seemed logical to search for a common cause for the variations in growth and the development of the skeleton. He suggested that both might be due to thyroid function. Holmgren's investigation deals with the time when the ossification process is coming to an end, and is therefore of less interest when the earliest stages of bone development in children are to be considered.

HASSELWANDER (1909) found a connection between height and ossification when studying the skeleton of the foot in a series of 521 subjects between the ages of 7 and 35. Like Holmgren, he thought an obvious conclusion to make was that both height and ossification are regulated from one and the same organ, such as the thyroid gland or possibly other glands furnishing an internal secretion. Like his predecessors, Hasselwander did not work up his material from the statistical standpoint and the value of his statements is therefore reduced in consequence.

In 1918, ÅKERLUND described an investigation on the skeleton which, unlike earlier publications, included several areas of ossification in the same material. A series of 28 boys and 28 girls between the ages of 8 and 14 years were selected with the greatest care, and weighed, measured, and roentgenographed for the purpose of studying the ossific centres in hand, wrist, elbow, and foot. The illustrations were arranged into groups according to the age, with a view to demonstrating the stage of development of the epiphyseal centres in question, during the particular period of growth. When comparing the height and the stage of ossification at different ages he found that in taller individuals there was greater differentiation in the skeletal development. His series of 56 subjects is, however, too small to give any final information on the connection between the development of bone and the height, and furthermore the investigation was concerned with a period when a great many of the bone centres have already appeared in the skeleton.

STETTNER published a report in 1921 on the results from a comprehensive investigation he had made on the development of the bones of the hand. The material consisted of 203 boys and 212 girls, 150 of the boys and 117 of the girls being 2 years of age or under, 35 boys and 46 girls between 2 and 6 years of age, and the remainder, 18 boys and 49 girls between 6 and 12 years of age.

He found a very extensive correlation between three factors, viz. age, height, and ossification. Earlier observers had stated that there was a direct proportion between increase in height and skeletal differentiation (von Wyss, Holmgren, Åkerlund, and others). Stettner found that this could not be the case except during a limited space of time at the close of the period of body growth and bone development, giving as a reason for this opinion the fact that if there was a continuous rate of growth tall individuals would reach full growth sooner than small ones. There must be some period during the years of growth when tall individuals cease to grow in height so that short individuals can catch up with them. He found that this cessation in growth occurred at some time between the age of 5 years and puberty.

In persons of small stature, on the other hand, growth was continuous, and they thus caught up with persons of large stature around the age of puberty. He observed exactly the same course of events in the ossification curve in the two groups. At 5 years of age there was a slowing down of the skeletal differentiation or even cessation of the ossification process in tall individuals, until, at puberty, they once again took the lead over smaller individuals who had displayed the whole time a continuous, although slow, skeletal differentiation.

Stettner produced no evidence to prove the correctness of these observations.

ADAIR and SCAMMON (1921) studied the ossific centres of the carpus in 100 newborn infants (45 boys and 55 girls). They found that there was a connection between body length and ossification, in that the centres were more differentiated in the longer infants. No statistical figures are quoted.

SAWTEL (1929) studied the relationships between height, weight, and bone development by means of correlation. Her material consisted of 112 children ranging in age up to 8 years. The skeleton of the hand only was studied. In comparisons between weight and height she found on the whole the same relation to the ossification. Her investigations also showed that only the ossification in the radius, metacarpal bones and phalanges was of any significance for an estimation of the skeletal development, and for these bones she obtained a correlation coefficient of $+0.32$ to $+0.51$. The ossification in the wrist was of little significance; possibly it was of some value for the examination of pathologic cases.

An objection that can be made against Sawtel's results is that her material of 112 children is too small to give statistical certainty, and it has in fact been found, when control investigations were made, that with her coefficient of correlation too large deviations are obtained.

In 1941, four American doctors, CHRISTIE, DUNHAM, JENSS, and DIPPEL, described an investigation on 1 107 newborn infants, carried out in order to study the ossific centre of the cuboid bone at birth. The material comprised 563 whites, of whom 297

were boys and 266 girls, and 544 negro children, 268 boys and 276 girls. With regard to the occurrence of the centre in infants of different lengths, it was found that the centre was present in 22 per cent of the infants less than 47 cm long, in 57 per cent of those measuring between 47 and 49 cm, and in infants measuring 50 cm or more there was an ossific centre in the cuboid in 71 per cent. No statistical treatment of the material was carried out.

No mention was made in the earliest publications of a connection between *body weight* and ossification. BEHRENDSEN (1897) wrote that children who were apparently sturdy were not always in advance of the weaker ones with respect to bone development, and VON RANKE (1898) reported that the state of nourishment seemed to play no part in the skeletal differentiation. HAHN (1899) thought he could establish that the skeleton was better developed in well-nourished children although he had also encountered cases where the weaker individuals had more ossific centres than those who appeared to be stronger. WILMS (1902) had found that weaker children sometimes even had better developed ossific centres.

All these investigations are limited and were carried out on small materials.

HASSELWANDER (1909) divided his material into different grades according to the state of nutrition of the individual. He was unable to find that the stronger children in this material displayed more active ossification, but he added, however, that it was not out of the question that a difference in ossification could exist between well-nourished and less well-nourished children. That he was unable to demonstrate any such difference was, in his opinion, due to the fact that his material was small and incomplete.

In his publication of 1918 ÅKERLUND reported that the more robust children in his series showed a better skeletal development; the best agreement was found between the degree of development of the skeleton and the bodily development when calculated according to BORNHARDT's (1886) formula, using the height, weight, and chest circumference. His material of 56 cases

is, however, too small to form the basis for a final conclusion in this matter. ÅKERLUND is the only one among the earlier observers who believed he had established a connection between the body's state of nourishment and the bony development.

Among the more recent publications dealing with the relation between weight and skeletal development mention may be made of SAWTEL's (1929). As already stated, she found, in her material, the same relation to the ossification for both height and weight. Her series of subjects is small, however, and later researchers consider that for this reason her results cannot be relied upon.

In the previously mentioned investigation by CHRISTIE, DUNHAM, JENSS, and DIPPEL (1941), on 1 107 infants, half of which were negro babies, the ossific centre in the cuboid was present in more of the infants of good weight than in those with a low weight. It was observed in 4 per cent of the infants weighing less than 2 500 g, in 34 per cent of those weighing 2 500—3 499 g, and in 49 per cent of those over 3 500 g.

The connection between *sex* and bony development had not been observed before the days of roentgenography, and the possibility of a difference between the sexes, with respect to ossification, was not mentioned in the first roentgenologic investigations on the skeleton of the hand published by BEHRENDSEN (1897) and VON RANKE (1898). VON WYSS (1899) shared the opinion of his predecessors, but he found that fusion between diaphysis and epiphysis, which marks the close of the period of growth, occurred earlier in women than in men. HAHN (1899) also was unable to see any difference between the sexes as regards the appearance of the ossific centres, but believed that fusion took place earlier in women. BADE (1899), from his studies of the skeleton of the foot, was inclined to believe that sex was of some significance, and stated that the ossification seemed to be more advanced in girls than in boys.

HOLMGREN (1909), in his afore-mentioned investigation, noted that ossification began earlier and that fusion also took place earlier in girls, and the same conclusion was reached by HASSEL-

WANDER (1910) in his studies of the development of the bones of the foot and by KÖHLER (1910) with regard to the elbow.

The ossific centres appear a few weeks earlier in girls even in the fetus (PRYOR, 1928). The latter author also found, like STETTNER in 1931, that the postfetal ossific centres in girls appear 3 to 9 months before those of boys, and according to the same author, fusion between epiphysis and diaphysis in girls can precede that of boys by as much as 3—4 years. No statistical calculations were made in any of these investigations.

As early as 1864, RAMBAUD and RENAULT mentioned the significance of *race differences* for the estimation of bone development. They had found in anatomic material a considerable difference in the epiphyseal centre at the proximal end of the tibia, in whites and negroes. Their material was very small, however, and no final conclusions can be drawn from it. In HAHN's investigation also, it was mentioned that when the proximal epiphyseal centre of the tibia was being studied a few negro children who showed more advanced ossification than the white children of the same age were included in the material. HASSELWANDER (1909) and FUJINAMI (1911) stressed the importance of race difference. STETTNER (1921) also mentioned this aspect, although he had made no particular investigation on it.

DOMMISSE and LEIPOLDT (1936) made an investigation with a view to studying the relation between skeletal development in different races. The material was composed of white and negro children, the wrist being the area examined. They found that the skeletal differentiation in the negro children was in advance of that in the whites of the same age.

CHRISTIE, DUNHAM, JENSS, and DIPPEL (1941) came to the same conclusion in connection with the previously mentioned investigation on the ossification of the cuboid bone in the newborn. In none of these publications is there any mention of statistical calculations. Nor are there any statements regarding the social conditions under which the different races lived. But as negroes, as is well known, often live under extremely straightened cir-

cumstances, it is conceivable that this factor also may have played some part.

The connection between other inherited qualities and the development of ossific centres has not, as far as I can find, been the subject of study. The inherited diseases will be discussed further on in another connection.

Even the first authors of the twentieth century discussed the influence of *environment* and *social conditions* on bone development. Thus, FUJINAMI (1911) considered the question of whether there is any difference in the development of the ossific centres in children from different social classes. After an investigation on the skeleton of the hand in 200 individuals from different classes of society he reported that he could see no differences with respect to ossification. His series of cases was so small, however, that it cannot be used as a basis for judgment. ÅKERLUND (1918), from the evidence of his afore-mentioned investigation, thought that a greater advance in the ossification could be observed in children from well-to-do homes than in those coming from families with lower incomes, but this observation was based on a few isolated cases and can hardly be of value for a final solution to the problem.

The first observer to study the problem in some detail was STETTNER (1931). He divided his material into middle-class children, working-class children, and country children, and found that the first-mentioned category showed the best skeletal differentiation, and the country children the worst, while the working-class children came somewhere in between the two. His investigation is so carefully laid out, and his material so uniform, that his findings must be attributed much significance, even though he did not examine the material from the statistical standpoint. Later investigators have made references to Stettner.

It is well-known that *climate* can affect bodily development. The literature furnishes no information as to whether this influence extends to skeletal development. RUCKENSTEINER (1931), however, states that a more rapid rate of ossification is said to have been observed at low latitudes than at high ones. Since growth and maturity proceed more rapidly in southern climes

it ought to be justifiable to assume that the same feature applies to the development of bone, and that Ruckensteiner's statement is correct, seeing that all authors are agreed that there is an intimate connection between the development of the skeleton and the bodily development as a whole.

No study has been made, either, on the development of ossific centres in relation to the different *seasons*, but according to MALLING-HANSEN (1883), NYLIN (1929), and others, there is a sure though slight variation in the bodily growth in relation to the seasons.

The question of pathologic disturbances in the development of the ossific centres will not be discussed in this thesis. It can be mentioned in passing, however, that a number of authors have established the existence of disturbances in connection with various affections of the organs of internal secretion. VON WYSS (1900), for instance, observed them when examining cretins, and BIRCHER (1909), HOLMGREN (1910), and JOSEFSON (1915) also mentioned them.

In recent years attempts have also been made to demonstrate that vitamins, in particular vitamin D, play a part in the skeletal development. It seems logical to assume that they do, since a deficiency of vitamin D is one of the causes of rickets.

Brief mention can be made here of MACARTHUR's (1936) investigations on the effect of deficiency diseases on bone development, FRANCIS's (1939) studies on the significance of vitamin D for epiphyseal skeletal maturity in children, and DANIEL's (1941) and WILBAR's (1942) investigations on the influence exercised by the vitamins A and D on the ossification process in the skeleton.

These investigations are built on small series, but they all point to the fact that there must be a connection between some of the deficiency diseases and ossification. Most authors have compiled tables for practical use, based on earlier publications, and mention can be made here of the ossification curves and tables published by SCHINTZ (1928), HASSELWANDER (1928), RUCKENSTEINER (1931), FLORY (1936), TODD (1938), and COFFEY (1945). In these tables, the lower and upper limits for the appearance of the individual epiphyseal centres have been established. The

most useful of these sets of tables are those of Ruckensteiner, since his ossification curves are so constructed as to indicate the lower borderline for the centres and at the same time show the date when the ossific centres are usually visualized on the roentgenogram. In America, similar sets of tables have been compiled by FLORY (1936) and by TODD (1938). Todd endeavoured to estimate the stage of development of the ossific centres by studying their density, setting up 9 different stages for the purpose. ROTCH constructed a similar table with 30 different stages of development. These tables are of little value for clinical purposes, as wide experience is required before they can be applied in practice. BAYER and NERWELL (1940) examined children for 2 years, using roentgenography with the object of studying the value of Todd's tables. They could find no agreement between these tables and their own findings. DANIELS (1941) also obtained similar results when examining Todd's standard tables.

In the recently published American text-book on diagnostic radiology, Pediatric X-Ray Diagnosis, (September 1945) COFFEY writes that the skeleton of the hand offers the most accessible and practical part on which to study skeletal development, and making use of VOGT's and VICHERS' (1938) tables he has worked out an ossification scheme from children of both sexes up to $6\,^1/_2$ years of age; from this age he follows the standard tables compiled by Todd and by Flory for the hand and wrist as well as for the foot.

These tables have about the same value as his predecessors', as they only deal with the ossification areas in the hand and foot, thus, with single ossification centres.

On account of the varying results obtained by earlier investigators in their attempts to determine by roentgenography the age of the skeleton and its relation to bodily growth in general, three American researchers decided to approach the problem from a different angle. The difficulty of obtaining a clear idea of the degree of development of the whole skeleton from the findings in one single area, due to the wide range of variability in the appearance of the individual ossific centres, had already been pointed out. SONTAG, SNELL and ANDERSON (1939) therefore

studied a number of ossification centres in one and the same material; they chose all the extremities on one side of the body. A material of 75 boys and 75 girls were roentgenographed at set intervals of 3 months up to the age of one year, then at six-month intervals up to the age of 5 years. All the ossific centres observed at the examination were added together and the figure obtained was assumed to be an arithmetical estimation of the skeletal development. The figures were then tabulated, the tables being intended for practical use. It is to be observed from these tables that the girls were well in advance of the boys the whole time, as regards the number of ossific centres, which means that their ossification proceeds more rapidly than that of the boys.

This investigation, taking in so many ossification areas simultaneously, must surely furnish a more accurate picture than the earlier studies of isolated areas. As has already been pointed out, however, age is not the only deciding factor for an estimation of the maturity of the ossific centres, and as such factors have not been taken into consideration in this publication the findings lose some of their value.

Summing up, it can be said that through the work of earlier investigators a more or less thorough knowledge has been gained regarding the development of the ossific centres in relation to age, and standard tables have been compiled. It has also been demonstrated that skeletal development is correlated not only to age but also to height, but nothing has been established regarding which of these correlations is the strongest.

The investigations have in the main been carried out with inadequate statistical methods. The extent to which environmental factors of various kinds influence development of bone has received little attention. The various circumstances relating to nutrition, which constitute, for instance, a part of the difference between the different social classes, have, however, not been determined, and such a study involves, of course, great difficulties. It has, on the other hand, been established that there is a difference in development between boys and girls. As differences in bone development can be expected to exist between different nations, not only because of race variations

but also on account of differing environmental factors, and in order to gain a clearer conception of the circumstances surrounding the appearance of the ossific centres, their correlation to age, and other such aspects, I have assembled and studied a series of Swedish children growing up under Swedish living conditions and in a Swedish environment. This material will be examined from the standpoint of some of the factors believed to affect the appearance of the ossific centres.

CHAPTER 2.

Material and Methods.

a. Material.

The material used in this investigation is composed of children from the city of Stockholm and the surrounding built-up areas, and was selected from among the children treated or examined at the Samariten Children's Hospital, Stockholm, during the years 1942—1945. There are 429 boys and 423 girls in the series, and 1 190 observations were made on these.

As the purpose of the investigation was to study the ossification process in absolutely healthy children of good physique and psychically well-developed, an anamnesis was obtained with regard to each child, special attention being paid to any disturbances in the organs of internal secretion that might have been present in the family. Information was also requested regarding the age of the mother when the child was born, and as to any disease in the mother, or other circumstances likely to have a bearing in this connection. The order of the child among its brothers and sisters, its age and its weight and height at birth were also ascertained.

In the selection of the material each child was given a physical examination and weighed and measured, and if nothing pathologic was observed it was then submitted to roentgen examination.

The child material thus studied belongs to the early years of childhood, the upper age limit having been set at 5 years because by that age all the ossific centres studied, with the exception of one, have begun to appear, and most of them are even fully developed.

The distribution among the different ages is shown in table 1. Many observations were made at short intervals in order to be

Table 1. Distribution of the observations among the different age groups.

Age group (months)	Number of		Age group (months)	Number of	
	Boys	Girls		Boys	Girls
1	45	48	13—15	26	28
2	37	37	16—18	28	26
3	32	25	19—21	15	20
4	24	21	22—24	23	21
5	28	19	25—27	22	19
6	26	22	28—30	16	23
7	21	17	31—33	22	18
8	22	22	34—36	15	18
9	20	23	37—42	21	31
10	19	22	43—48	27	24
11	16	20	49—54	20	19
12	19	19	55—60	24	21

able to determine with as high a degree of certainty as possible the date at which each ossific centre appears.

In the different observation groups, the time for each examination was placed as close as possible to the indicated month figures, and during one year a maximum of 5 days from these figures was allowed. After one year a maximum of 10 days was allowed. Later on, when the material was being worked up, it was found more practical consistently to take the age figures as the class midpoints in each group.

In this way, a study was made not only of the bone development in the extremities of the right side, at 1 131 observations, but also of that on the left side, at 59 observations carried out concurrently for the purpose of comparing the skeletal differentiation in the two halves of the body. The distribution of these 59 cases on different age groups is shown in table 2.

It is of great importance to determine the degree of maturity at birth, since it is conceivable that the development may be different in prematurely born children. If possible, the material ought not to include any children except those born at term.

Table 2. No. of observations made simultaneously on right and left side.

Age group (months)	Number of		Age group (months)	Number of	
	Boys	Girls		Boys	Girls
4	—	2	22—24	1	—
5	2	—	25—27	1	2
6	1	4	28—30	1	2
7	1	—	31—33	1	—
9	—	1	34—36	1	3
10	4	2	37—42	3	3
11	1	1	43—48	3	5
13—15	2	1	49—54	1	1
16—18	1	3	55—60	4	—
19—21	—	1			

As the object of the investigation was to make it possible for normal tables to be compiled for practical use the borderlines should not be drawn too strictly, however. When a child is presented for examination it is not always possible to obtain reliable information regarding the circumstances connected with the birth. The values which we use as a basis should be obtained from »normal material» if they are to be used on normal material.

Table 3. Weight and height at birth.

$M \pm \varepsilon(M)$ = mean ± standard error of the mean. σ = standard deviation.

	Boys			Girls		
	Number	$M \pm \varepsilon(M)$	σ	Number	$M \pm \varepsilon(M)$	σ
The author's material						
Weight, gm	569	3297 ± 29	695	562	3098 ± 32	779
Height, cm	568	50.5 ± 0.14	3.2	560	49.0 ± 0.20	4.7
Material from Linders						
Weight, gm	8675	3480 ± 5	480	8470	3350 ± 5	450
Height, cm	8661	50.9 ± 0.02	2.1	8459	50.2 ± 0.02	2.1

In order to investigate how the matter stands in my material I made a comparison with figures given by LINDERS (1930). It was found that with respect to weight the figures for my material of boys and girls were slightly lower. It was also found that the standard deviation was somewhat higher. As regards height, the relations were approximately the same, for both boys and girls. As to the mean, there was no essential difference in height. The standard deviation, on the other hand, was slightly larger in my material. On the whole, these divergences cannot be said to be of much significance. One might take it that my material corresponds in the main to LINDERS' normal material with a pregnancy time of 40 weeks. The standard deviations, however, are larger throughout in my material, a fact which may indicate that there is greater variation, and that there are children born after a pregnancy time of more or less than 40 weeks in my material. (Table 3.)

b. Methods.

Weighing, and measuring of the height formed part of the physical examination. The babies were weighed on baby scales and the larger children on a normal-sized weighing instrument. All the children were weighed naked. The height was measured by means of a specially constructed yardstick fitted with a steel measure graduated in millimetres.

For the roentgen examination, an anteroposterior view was taken of each ossification area, with the exception of the knee-joint, which was photographed from the side so as to avoid the patella. As it is very difficult to photograph the fingers and toes of infants the method used was to pin the hand and foot to the underlying support by means of a cushion and hold a wooden spade on the cushion.

All the bones of the extremities on the right side were roentgenographed, i. e. the hand, wrist, elbow, and shoulder, foot, ankle, knee, and hip. For purposes of comparison, the bones of the extremities on the left side also were roentgenographed at 59 observations.

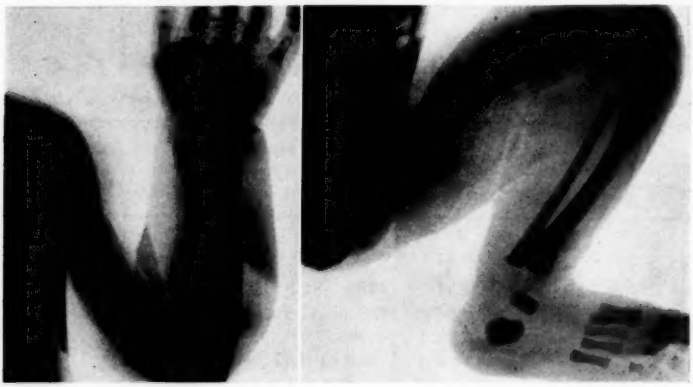


Fig. 1. Roentgen examination of young infants with the tubes in two positions, one for the upper limbs and one for the lower limbs.

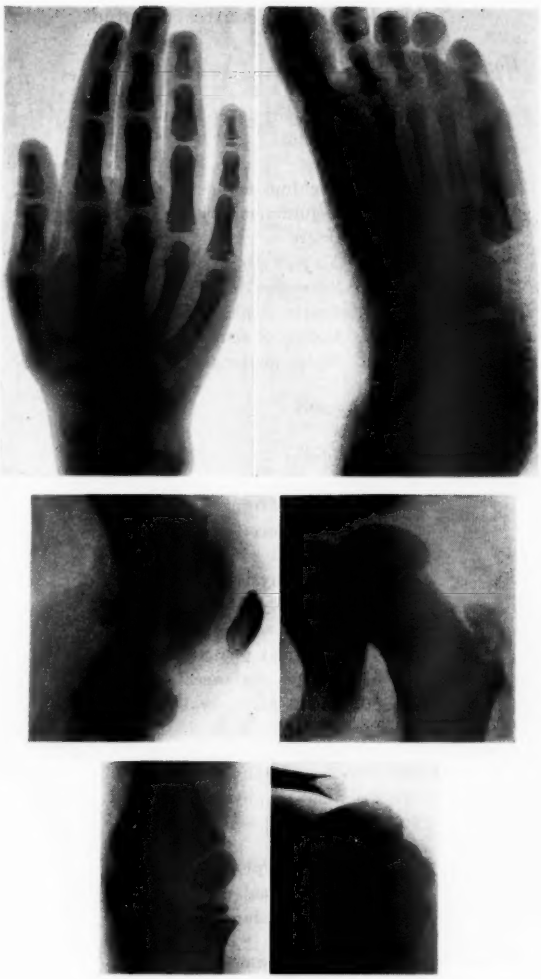


Fig. 2. Roentgen examination of larger children with the tubes in six different positions, one for each area of ossification.

Table 4. List of centres counted.

Hand: No. 1. Capitatum
2. Hamatum
3. Distal epiphysis of radius
4. Triquetrum
5. Lunatum
6. Multangulum majus
7. Multangulum minus
8. Naviculare
9. Distal epiphysis of ulna
10—14. Metacarpal epiphyses
15—19. Proximal phalangeal epiphyses
20—23. Middle phalangeal epiphyses
24—28. Distal phalangeal epiphyses

Foot: 29. Calcaneus
30. Talus
31. Cuboideum
32. Third cuneiform
33. Second cuneiform
34. First cuneiform
35. Naviculare
36. Distal epiphysis of tibia
37. Distal epiphysis of fibula
38—42. Metatarsal epiphyses
43—47. Proximal phalangeal epiphyses
48—51. Middle phalangeal epiphyses
52—56. Distal phalangeal epiphyses

Knee: 57. Distal epiphysis of femur
58. Proximal epiphysis of tibia
59. Proximal epiphysis of fibula
60. Patella

Hip: 61. Proximal epiphysis of femur
62. Trochanter major
63. Trochanter minor

Elbow: 64. Capitulum humeri
65. Proximal epiphysis of radius

Shoulder: 66. Proximal medial epiphysis of humerus
67. Proximal lateral epiphysis of humerus
68. Proc. coracoideus

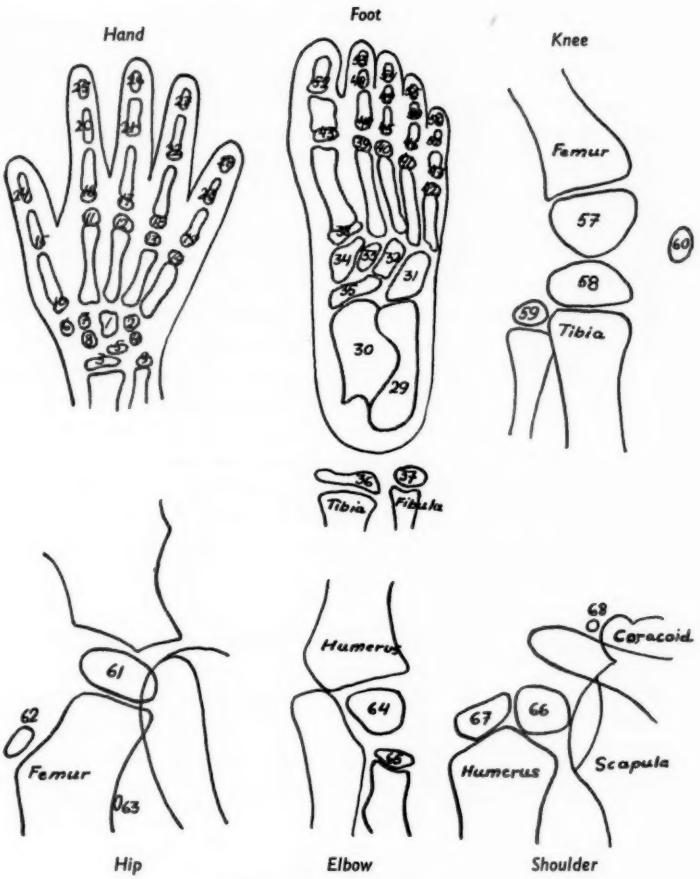


Fig. 3. Centres counted.

By using the least possible of film-surface for each ossification area the whole series of pictures could be contained in 3 or 4 roentgen films 13 × 18 cm in size. For the young infants, two films were sufficient in most cases, one for the upper and one for the lower extremities. Figures 1 and 2 illustrate the method of examination.

The exposure times were specially worked out for the roentgen apparatus of the hospital, varying between 0.06 and 0.3 seconds according to age and size. In all, 4 000 roentgen films were used for the 1 131 observations on the bone development in the right half of the skeleton and the 59 observations made on the left half. A total of 7 100 roentgen pictures were made.

The whole examination took only a few minutes to perform on each occasion.

In the interpretation of the films, the ossific centres were recorded as soon as they produced a distinct shadow on the plate, irrespective of what their size was. Any accessory ossific centres that happened to be present were not recorded.

In order to facilitate the study of the films, and to make the material easier to handle for the mathematical calculations the ossific centres were numbered as shown in table 4 and figure 3.

In the statistical treatment of the material the commonly employed methods were used (see DAHLBERG, 1940). When more unusual methods were used attention is drawn to them in the text.

CHAPTER 3.

Relation between Ossification and Age.

The term degree of maturity is often applied to children without there being any clear idea as to what is meant. In all probability, what is vaguely referred to is the differentiating of the tissues. It is not likely that this differentiation runs parallel in all respects. In the aging process, as we know, the changes are not always parallel and harmonious.

On the whole, however, we can assume that the differentiation process proceeds uniformly, and that if one tissue is developed and differentiated others will also be differentiated. From this point of view, the development of the ossific centres can be assumed to serve as a good guide to the degree of differentiation. This differentiation thus ought to proceed parallel to, for instance, age and height. Before any clear conception can be gained on this question, however, it is necessary to examine an adequately large material.

Two statistical methods can be used for ascertaining how the ossific centres develop. One is to follow the bone development taking place in a series of individuals as they grow older. Here, it is the same individual whose ossific centres are examined at different ages. The other method is to determine the stage of the bone development in different individuals of different ages. With this method it is not a matter of following special individuals. The two methods ought, of course, to give the same result, if the material is large enough. With the same number of determinations, more individuals will be included in the series if the second method is used, and the material will thus be more representative of the population, but otherwise the method chosen is probably

immaterial. In the present series of 429 boys and 423 girls, i. e. 852 individuals, 1 131 observations were made, 568 on boys and 563 on girls. As can be seen, examinations were made more than once only in a few cases, and the material can thus be said to belong to the latter type.

The information obtained can be worked up in two different ways. One way is to show how many individuals have a particular ossific centre at different ages. An s-shaped curve will then be obtained. The date of appearance of the centre can be calculated from the curve by successive subtraction. The curve thus obtained ought to resemble in the main a probability curve. A schematic representation of the curves is shown graphically in figures 4 and 5. In both ways the development of the ossific centres can be determined by means of quartiles and medians, and this is the method employed in the present investigation.

Before describing the method by which the ossific centres were studied in this material the development of the centres during embryonic life will be briefly surveyed, the description supplied by BROMAN (1928) being used as a basis.

Embryonic differentiation of the skeleton begins early. Even in embryos of 10 mm there is an accumulation of blastema within the forelimb buds, and shortly afterwards similar bone-rudiments appear in the hind-limb buds.

The upper limb-rudiments then continue to develop rapidly through the appearance of a mesodermal core in the region of the upper humerus, the proximal segment of which increases in length towards the scapula region while the distal segment grows in the direction of the hand-plate. The ossification in the arm begins in embryos of 15 mm with the appearance of a centre in the clavicle-rudiment, and shortly after this the bone-rudiments of the rest of the arm appear. In the phalanges and metacarpal bones, the primary centres begin to appear in 30 mm long embryos, the second phalanx of the thumb being the first to form and the second phalanx of the little finger the last, in embryos of 12 cm. All the epiphyses of both carpal bones and arm remain at the cartilage stage during the whole of the intra-uterine period. The ossification of the skeleton of the lower limbs starts in the

femur and tibia, in embryos of 18—20 mm. In the phalanges and metatarsal bones, the process starts in the second phalanx of the great toe and the second metatarsal bone, and ends with the

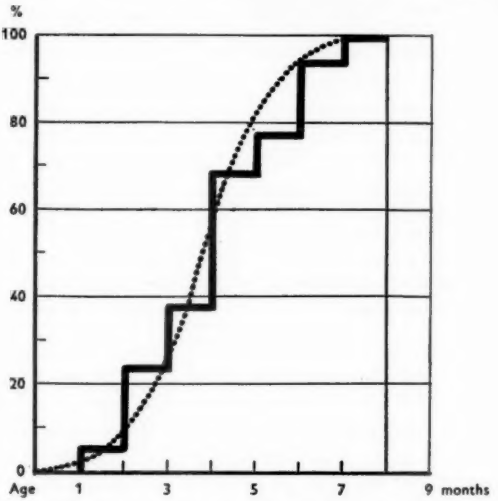


Fig. 4. Curve showing the number of ossific centre No. 61 in different months.

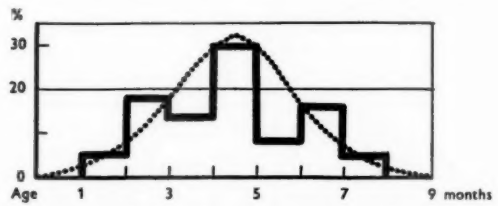


Fig. 5. Probability curve obtained by successive subtracting from the distribution in Fig. 4 and showing the development of the ossific center No. 61 during different months.

second phalanx of the little toe, in the ninth month of fetal life. The two largest tarsal bones, the calcaneus and the talus, already have their centres by the sixth month of fetal life and the centre for the cuboideum appears shortly before or after birth. The rudiments of the other tarsal bones consist of cartilage at birth.

Table 5 a. Age in months, when the ossific centres of the hand appear

List of ossific centres counted	Boys				
	First appearance	25 %	50 %	75 %	100 %
1. Capitatum	0	3.6	4.2	4.9	9.0
2. Hamatum	0	4.0	5.0	7.0	10.0
3. Distal epiphysis of radius	4	7.7	10.4	13.1	15.0
4. Triquetrum	5	21.1	32.0	45.6	51.5
5. Lunatum	7	36.7	49.8	57.8	—
6. Multangulum majus	28—30	53.1	—	—	—
7. Multangulum minus	43—48	57.8	—	—	—
8. Naviculare	37—42	—	—	—	—
9. Distal epiphysis of ulna	49—54	—	—	—	—
10. Metacarpal epiphysis I	16—18	25.5	31.4	34.0	—
11. » » II	7	17.4	22.9	25.1	35.0
12. » » III	12	17.0	23.3	26.5	35.0
13. » » IV	13—15	22.0	24.9	29.4	35.0
14. » » V	13—15	23.0	26.1	29.6	51.5
15. Proximal phalangeal epiphysis I	11	26.6	31.4	33.3	57.5
16. » » » II	7	12.3	17.1	21.7	26.0
17. » » » III	7	12.4	14.0	17.0	29.0
18. » » » IV	7	12.4	16.3	23.1	29.0
19. » » » V	7	17.5	23.5	25.7	29.0
20. Middle » » I	12	17.3	24.2	26.3	45.5
21. » » » II	10	17.0	23.1	25.7	45.5
22. » » » III	10	17.0	22.9	25.6	45.5
23. » » » IV	13	31.4	34.4	44.8	—
24. Distal » » I	7	13.0	16.3	23.6	35.0
25. » » » II	16—18	31.9	34.4	43.1	51.5
26. » » » III	12	22.6	28.2	31.4	51.5
27. » » » IV	13—15	22.8	29.3	32.7	51.5
28. » » » V	13—15	32.1	34.8	44.6	51.5

All the epiphyseal centres in the limbs appear after birth, with the exception of those for the distal end of the femur and the proximal end of the tibia, which appear shortly before birth, usually in the tenth month of intra-uterine life but very often also after birth.

The present investigation was concerned only with the demonstration of ossific centres by roentgenography. A centre

and when they are present in 25, 50, 75 and 100 per cent of the cases.

List of ossific centres counted	Girls				
	First appearance	25 %	50 %	75 %	100 %
1. Capitatum	0	1.8	3.2	4.7	7.0
2. Hamatum	0	2.7	4.0	5.3	8.0
3. Distal epiphysis of radius	5	7.7	8.0	12.1	20.0
4. Triquetrum	7	22.2	27.8	34.4	51.5
5. Lunatum	13—15	29.8	35.1	50.4	—
6. Multangulum majus	28—30	43.1	55.2	—	—
7. Multangulum minus	28—30	47.0	56.8	—	—
8. Naviculare	28—30	46.5	59.8	—	—
9. Distal epiphysis of ulna	25—27	57.9	—	—	—
10. Metacarpal epiphysis I	11	16.1	19.8	22.7	32.0
11. » » II	10	11.7	13.1	18.8	23.0
12. » » III	10	13.0	15.9	18.8	23.0
13. » » IV	11	14.0	17.2	19.9	23.0
14. » » V	11	14.9	17.0	21.0	32.0
15. Proximal phalangeal epiphysis I	8	17.5	20.3	23.4	32.0
16. » » » II	8	10.2	11.1	14.4	20.0
17. » » » III	8	10.1	11.0	13.6	20.0
18. » » » IV	8	10.2	11.3	14.7	20.0
19. » » » V	10	12.8	14.5	18.0	26.0
20. Middle » » I	11	12.4	16.0	19.3	26.0
21. » » » II	10	11.8	14.9	18.8	26.0
22. » » » III	10	12.1	14.9	19.3	26.0
23. » » » IV	9	18.9	22.2	25.9	35.0
24. Distal » » I	8	8.8	11.0	17.5	26.0
25. » » » II	11	19.8	22.9	27.7	35.0
26. » » » III	11	12.5	20.3	23.0	32.0
27. » » » IV	11	14.0	20.3	24.3	32.0
28. » » » V	11	19.9	22.9	26.3	35.0

can only be expected to appear on a roentgenogram when it has attained a certain size, and the dates of appearance obtained by roentgen examination are therefore later than those obtained at an anatomic examination.

In tables 5 a, 5 b, and 5 c we have a survey of the dates of appearance of the ossific centres. Medians and quartiles are indicated in the tables as well as the date of the first appearance

Table 5 b. Age in months, when the ossific centres of the foot appear and

List of ossific centres counted	Boys				
	First appearance	25 %	50 %	75 %	100 %
29. Calcaneus	0	0	0	0	0
30. Talus	0	0	0	0	0
31. Cuboideum	0	0.8	2.0	3.7	7.0
32. Third cuneiform	0	4.1	5.0	7.4	20.0
33. Second cuneiform	7	19.0	25.5	38.3	51.5
34. First cuneiform	7	23.6	31.4	36.3	51.5
35. Naviculare	12	31.6	38.1	51.5	—
36. Distal epiphysis of tibia	1	3.9	5.7	6.7	10.0
37. » » » fibula	6	9.7	10.8	22.5	35.0
38. Metatarsal epiphysis I	22—24	28.3	31.9	33.8	35.0
39. » » II	28—30	34.8	39.0	45.2	51.5
40. » » III	28—30	37.5	39.5	49.0	57.5
41. » » IV	28—30	42.9	47.5	51.5	—
42. » » V	28—30	43.7	48.2	55.7	—
43. Proximal phalangeal epiphysis I	9	24.3	28.8	32.7	45.5
44. » » » II	13—15	17.2	21.9	25.8	29.0
45. » » » III	13—15	14.7	20.8	23.4	29.0
46. » » » IV	13—15	16.0	21.8	22.4	29.0
47. » » » V	16—18	23.5	28.8	32.7	45.5
48. Middle » » I	13—15	21.7	24.3	28.6	35.0
49. » » » II	10	15.2	25.3	29.0	35.0
50. » » » III	9	17.7	26.2	35.0	—
51. » » » IV	9	29.0	35.1	47.4	—
52. Distal » » I	8	12.9	14.0	19.6	26.0
53. » » » II	13—15	34.2	39.5	47.3	—
54. » » » III	16—18	35.2	41.5	47.1	—
55. » » » IV	16—18	37.1	41.7	46.8	—
56. » » » V	9	36.7	39.5	49.5	—

of the centres and the date when all the centres are present. It is obvious that the last-mentioned dates must be ascribed to chance.

As will be seen from the tables, the distribution seems to be skew for the dates of appearance of many of the centres. The first and third quartiles differ from one another. This can be due to chance, as the material is not very large. It is not sufficiently large to enable us to calculate the skewness in the

and when they are present in 25, 50, 75 and 100 per cent of the cases.

List of ossific centres counted	Girls				
	First appearance	25 %	50 %	75 %	100 %
29. Calcaneus	0	0	0	0	0
30. Talus	0	0	0	0	0
31. Cuboideum	0	0.6	1.7	2.5	5.0
32. Third cuneiform	0	2.7	4.2	5.3	9.0
33. Second cuneiform	8	11.4	20.6	24.3	32.0
34. First cuneiform	9	20.1	22.5	25.4	35.0
35. Naviculare	13—15	22.6	25.9	35.1	51.5
36. Distal epiphysis of tibia	3	4.7	5.6	6.6	7.0
37. » » » fibula	1	7.3	8.8	10.7	20.0
38. Metatarsal epiphysis I	8	17.8	20.0	22.2	26.0
39. » » II	13—15	23.1	25.9	28.6	32.0
40. » » III	19—21	25.5	29.1	32.0	35.0
41. » » IV	19—21	28.4	32.0	35.9	51.5
42. » » V	19—21	32.1	35.0	43.0	57.5
43. Proximal phalangeal epiphysis I	8	17.2	19.6	20.0	29.0
44. » » » II	8	11.0	14.9	18.8	23.0
45. » » » III	8	10.9	12.3	16.8	23.0
46. » » » IV	8	11.0	13.3	19.3	23.0
47. » » » V	8	18.0	21.0	23.0	26.0
48. Middle » » I	10	11.0	16.9	20.0	26.0
49. » » » II	10	11.2	17.5	23.4	—
50. » » » III	10	12.4	22.4	26.3	—
51. » » » IV	12	20.0	25.9	35.7	—
52. Distal » » I	8	9.1	10.2	13.2	26.0
53. » » » II	10	22.4	29.1	33.0	57.5
54. » » » III	13—15	20.0	29.3	34.4	51.5
55. » » » IV	13—15	22.6	29.5	35.4	51.5
56. » » » V	12	23.1	30.1	36.0	51.5

ordinary way. We have therefore confined ourselves to calculating the skewness, according to LINDEBERG's method, for some of the centres in respect of which the distribution seems to be especially skew. The calculation refers to centres nos. 11, 25, and 52. The result is given in table 6.

It will be observed that the skewness is not statistically significant for any of these ossific centres. In any case, the

Table 5 c. Age in months, when the ossific centres of the knee, the hip, in 25, 50, 75 and 100 per

List of ossific centres counted	Boys				
	First appearance	25 %	50 %	75 %	100 %
Knee					
57. Distal epiphysis of femur	0	0.3	0.5	0.8	3.0
58. Proximal epiphysis of tibia . . .	0	0.4	0.7	1.8	4.0
59. » » » fibula . . .	31—33	40.8	51.3	56.4	—
60. Patella	34—36	43.8	49.0	55.9	—
Hip					
61. Proximal epiphysis of femur . . .	1	3.9	5.6	6.7	10.0
62. Greater trochanter	28—30	43.2	46.1	51.5	—
63. Lesser »	—	—	—	—	—
Elbow					
64. Capitulum humeri	1	3.7	6.0	8.7	26.0
65. Proximal epiphysis of radius . . .	13—15	47.5	53.3	—	—
Shoulder					
66. Proximal medial epiphys. of humer.	0	0.7	2.1	3.6	6.0
67. » lateral » » »	4	9.9	19.5	23.6	—
68. Proc. coracoideus	1	3.7	5.0	10.1	20.0

skewness is due in part to chance irregularities, and is partly to be explained by the fact that the observations are not exact.

In order to gain a general idea of the situation, the skewness for all the centres has been calculated by comparing the first quartile with the third. In other words the relation between these has been calculated. If this number is 1, the distribution is symmetrical, if it is below 1 the third quartile is larger than the first; if it is more than 1 the first quartile is larger than the third. It will be found that for the boys the quotient is below 1 for 26 ossific centres and above 1 for 33. For the girls, there are 38 centres with a quotient of over 1 and 21 with a quotient below 1. The mean for boys will be 1.53 ± 0.21, and 1.59 ± 0.23 for girls. Despite the fact that so many centres were examined, the deviation from 1 is not statistically significant but can instead

hip, the elbow and the shoulder appear and when they are present per cent of the cases.

List of ossific centres counted	Girls				
	First appearance	25 %	50 %	75 %	100 %
Knee					
57. Distal epiphysis of femur	0	0.3	0.6	0.9	5.0
58. Proximal epiphysis of tibia . . .	0	0.4	0.8	2.3	5.0
59. » » » fibula . . .	22—24	30.1	34.5	43.5	57.5
60. Patella	22—24	27.3	31.6	33.3	51.5
Hip					
61. Proximal epiphysis of femur . . .	2	3.6	4.9	6.2	8.0
62. Greater trochanter	19—21	27.2	30.2	34.0	51.5
63. Lesser »	—	—	—	—	—
Elbow					
64. Capitulum humeri	1	4.3	5.7	8.0	11.0
65. Proximal epiphysis of radius . . .	22—24	34.6	44.2	47.9	—
Shoulder					
66. Proximal medial epiphys. of humer.	0	0.7	2.1	3.8	6.0
67. » lateral » » »	4	7.7	9.1	10.7	32.0
68. Proc. coracoideus	1	5.6	6.4	7.3	17.0

be due to chance. There is thus no reason to assume that the distribution in general must be skew in reality, but for safety's sake we used the median and quartiles, and not the mean, in our study of the appearance of the ossific centres.

In figures 6—11 will be found a graphic presentation of the medians and quartiles of the centres as a complement to tables 5 a, 5 b, and 5 c.

As a result of the information provided by these tables and figures, the following statements can be made regarding the dates of appearance of the ossific centres.

In the ossification area of the wrist and hand, in *boys*, the ossific centres for the capitatum and hamatum appear before the age of one month. In addition to the distal epiphysis of the radius, which begins to appear at 4 months of age, these two ossific centres

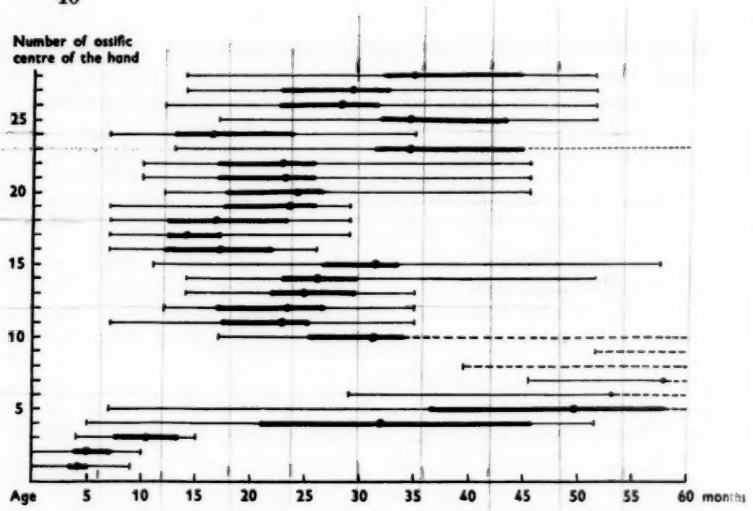


Fig. 6. Age in months when the ossific centres of the hand in boys appear. The horizontal lines show the range of variation for the appearance of different ossific centres. (Number of the centres is given to the left). On the lines, the median is marked with a point, the distance between the quartiles and the median with thick lines. The distance between the end of the variation and the quartiles is shown with a thin line. In some cases the ossific centres do not reach 100 per cent of the cases; the line is then broken.

Table 6. Skewness in the distribution according to Lindeberg.

$S_L \pm \varepsilon(S_L)$ = skewness according to Lindeberg ± standard error of the skewness.

Number of ossific centre	Number of observations	$S_L \pm \varepsilon(S_L)$ in per cent
11	8	+ 1 (± 11)
25	5	−11 (± 13)
52	9	− 7 (± 10)

form a group the entire development of which is concentrated to the first year of life. The epiphyseal centre in the radius, however, is not present in all children before the age of 15 months. The ossific centre for the triquetrum, which follows at 5 months

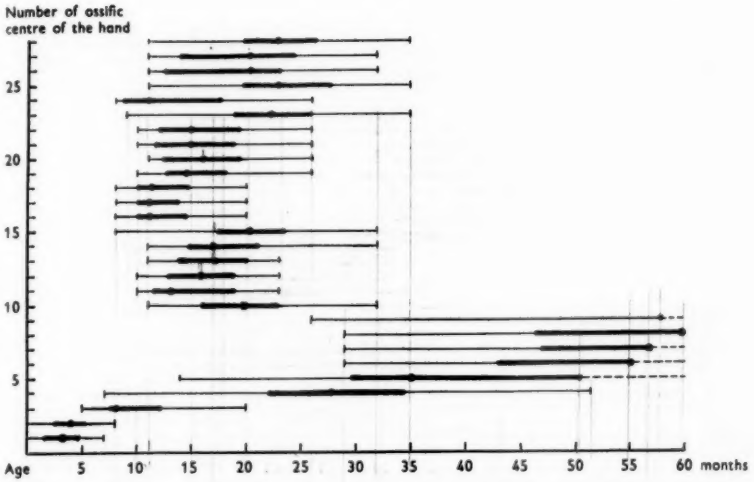


Fig. 7. Age in months when the ossific centres of the hand in girls appear. The horizontal lines show the range of variation for the appearance of different ossific centres. (Number of the centres is given to the left). On the lines, the median is marked with a point, the distance between the quartiles and the median with thick lines. The distance between the end of the variation and the quartiles is shown with a thin line. In some cases the ossific centres do not reach 100 per cent of the cases; the line is then broken.

of age, is not present in 50 per cent of the individuals until the age of 32 months, and not until the age of 51.5 months is it present in all; it thus covers a development period of 46.5 months. Still greater variability is displayed by the lunatum centre, which begins to appear at 7 months of age but is not even found in all at the age of 5 years. Of the other centres in the wrist, none appear until after $2\,^1/_2$ years of age, when the multangulum majus begins to develop its centre at the age of 29 months. The ossific centres for the multangulum minus and the naviculare, and the epiphyseal centre of the distal end of the ulna, only begin to appear during the last year, and at 5 years they have not reached 25 per cent of the individuals observed.

The ossification in the wrist is thus characterized by a division into two periods, the first one during the first year of life and a

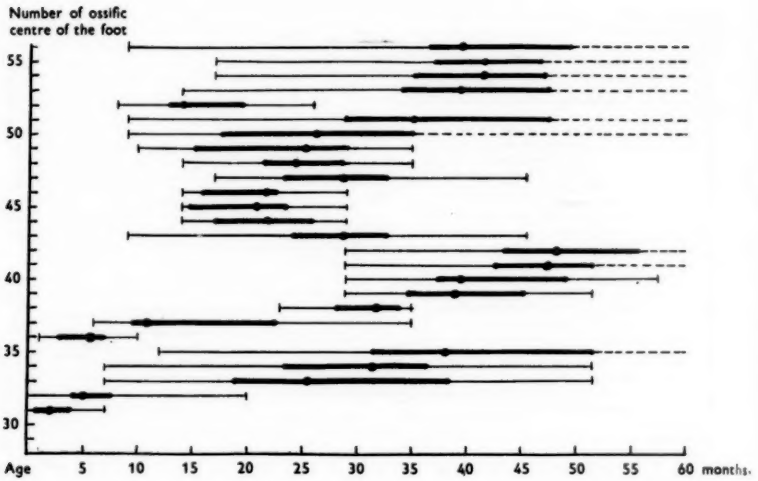


Fig. 8. Age in months when the ossific centres of the foot in boys appear. The horizontal lines show the range of variation for the appearance of different ossific centres. (Number of the centres is given to the left.) On the lines, the median is marked with a point, the distance between the quartiles and the median with thick lines. The distance between the end of the variation and the quartiles is shown with a thin line. In some cases the ossific centres do not reach 100 per cent of the cases; the line is then broken.

second one which begins shortly before the end of the first 5 year period.

Regarding the metacarpal bones, the variability for I and V is much greater than for the intervening bones, II and IV. While the centres for the first and last metacarpal bones cover a development period of about 36 months the corresponding time for the centres of the intervening bones is about 20 months. With the exception of the first metacarpal bone, which begins to appear later, the centres for the other metacarpal bones are visible on roentgenograms from the age of 7 months and onwards. The second metacarpal bone gets its centre first and is followed by the others at intervals of 2—5 months. The fourth and fifth metacarpal centres appear at the same time, however. In the quartile and median values of these centres it is possible to detect

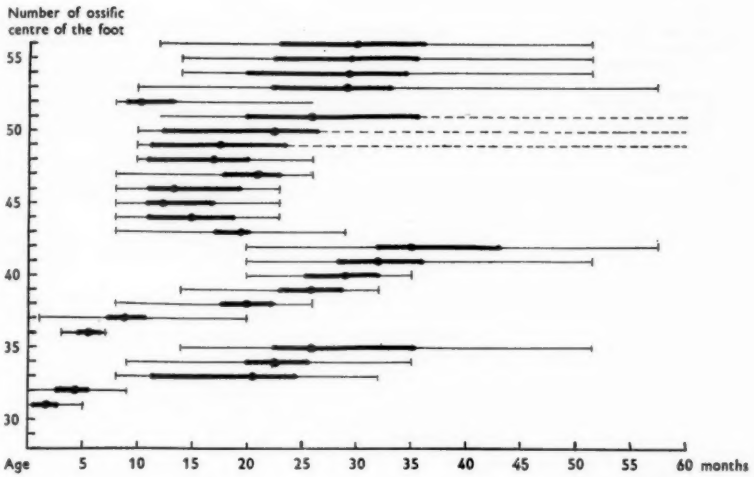


Fig. 9. Age in months when the ossific centres of the foot in girls appear. The horizontal lines show the range of variation for the appearance of different ossific centres. (Number of the centres is given to the left.) On the lines, the median is marked with a point, the distance between the quartiles and the median with thick lines. The distance between the end of the variation and the quartiles is shown with a thin line. In some cases the ossific centres do not reach 100 per cent of the cases; the line is then broken.

a certain »progression» in a radio-ulnar direction, which would seem to be an indication of the fact that the metacarpal bones, with the exception of the first, get their centres in a definite order.

As in the case of the metacarpal bones, the centres in the phalanges also to a certain extent show signs of appearing in groups. Thus, the proximal phalangeal epiphyses appear together, reach median and quartile values simultaneously, and are observed in all individuals at approximately the same date. The first phalanx forms an exception here also. This begins to appear later and is not present in all until after a period of nearly 4 years. A development in a radio-ulnar direction such as was observed in the metacarpal bones is not seen in the proximal phalanges.

The development of the four middle phalanges is, on the whole,

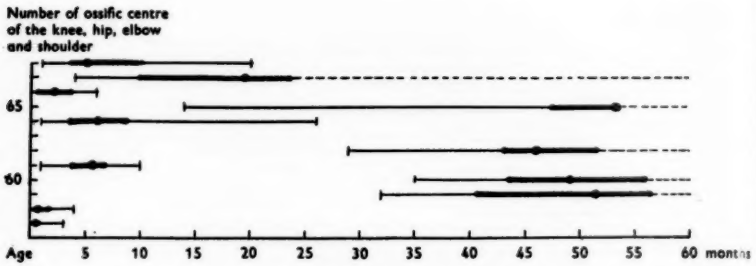


Fig. 10. Age in months when the ossific centres of the knee, hip, elbow and shoulder in boys appear. The horizontal lines show the range of variation for the appearance of different ossific centres. (Number of the centres is given to the left.) On the lines, the median is marked with a point, the distance between the quartiles and the median with thick lines. The distance between the end of the variation and the quartiles is shown with a thin line. In some cases the ossific centres do not reach 100 per cent of the cases; the line is then broken.

uniform. They begin to get their ossific centres at the same time and are present in all instances at the age of 45.5 months, thus having a development period covering 3 years. The last middle phalanx, however, is not present in all at 5 years of age. This phalanx is also the last of the bones of the hand to form during the embryonic stage, while the terminal phalanx of the thumb forms first. The ossific centre for the last-named phalanx also appears much earlier than those for the other phalanges, and it follows in the main the development of the metacarpal bones. The other terminal phalanges begin to ossify in no special order, and from one year of age, when they make their first appearance, to 51 months, when they are found in all boys, they display as great a variability as the centres for the middle phalanges.

In the elbow joint, the centres for the capitulum humeri and the proximal epiphysis of the radius were studied. It is strange that the ossific centre for the capitulum humeri should not be present in all boys before the age of 2 years although it begins to appear as early as the first month of life and has reached the third quartile within the first year. This value may possibly be ascribed to chance irregularities, as there was no similar feature among the girls. The centre for the proximal end of the radius

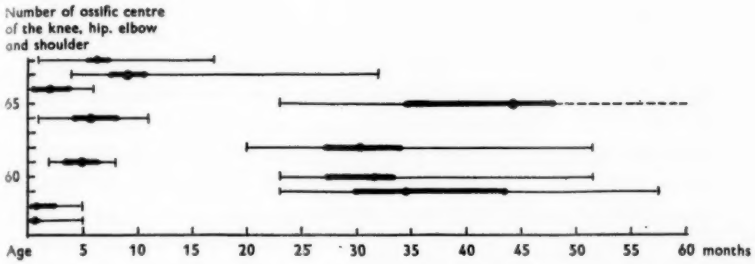


Fig. 11. Age in months when the ossific centres of the knee, hip, elbow and shoulder in girls appear. The horizontal lines show the range of variation for the appearance of different ossific centres. (Number of the centres is given to the left.) On the lines, the median is marked with a point, the distance between the quartiles and the median with thick lines. The distance between the end of the variation and the quartiles is shown with a thin line. In some cases the ossific centres do not reach 100 per cent of the cases; the line is then broken.

has a long development period, being represented in only 50 per cent of the children at 5 years of age despite the fact that it begins to appear shortly after the first year of life.

In the ossification area of the shoulder-joint three ossific centres, the medial and lateral centres for the head of the humerus and the centre for the coracoid process, were examined. Of these, the medial one has already appeared before the age of one month and is present in all cases at 6 months, while the lateral one appears at 4 months and is not present in all at 5 years. The coracoideus centre develops chiefly during the first year of life.

As regards the ossification in the lower limbs, the development seems on the whole to proceed along the same lines as in the upper limbs. The variability is also equally great.

In the ankle, the ossific centres for the talus and calcaneus are already present at birth, and during the first months of life the centres for the cuboideum and the distal epiphysis of the tibia also begin to ossify, being present in all cases at 7 and 10 months, respectively. The centre next in order of development, the third cuneiform bone, first appears before the age of 1 month, and reaches the third quartile at 7 months, but it is not found in all before the age of 20 months. The next centre does not appear

until six months later. The epiphyseal centre for the distal end of the fibula then begins to appear, although it has such a large variability that it is not found in all individuals until $2\,^1/_2$ years later. At 7 months, the next centres appear in the foot, namely the first and second cuneiform bones; of all the bones in the foot these have the greatest variability, 44.5 months. Possibly the os naviculare has a longer period of development; its centre first appears at 12 months but is not present in more than 75 per cent at 5 years.

The metatarsal bones do not appear until at $2\,^1/_2$ years of age; the only exception is the first metatarsal bone, in which the centre appears earlier and develops more rapidly. This is just the opposite to the corresponding bone in the hand, which develops later than the other metacarpal bones. The centres for the other metatarsal bones form and develop in the order from II to IV. They begin to appear, it is true, at approximately the same time, but their median and quartile values increase with the number of the centre in the same way as was observed in the metacarpal bones of the wrist. Their variability lies around two years, in this respect also they resemble the metacarpal bones.

The ossific centres for the proximal phalanges appear simultaneously, and it can on the whole be said that they keep pace with one another in development, although not to the same extent as the corresponding bones in the hand. The variability of the first and last phalanges is considerably larger than that of the intervening phalanges, being almost 2 times as large. Regarding the middle and terminal phalanges it will only be mentioned that the variability for both groups is large and that no mutual relation in development can be observed among them. As in the case of the hand, the ossific centre for the first terminal phalanx appears long before that of the others and is the only one which is present in all individuals at 5 years of age. In embryonic life also, this terminal phalanx is the first to form among the bones in the foot.

Of the ossific centres in the knee-joint, two, those for the distal epiphysis of the femur and the proximal epiphysis of the tibia, develop before the age of six months, and as was mentioned

further back, it very often happens that they are present at birth. In the present investigation they were in most cases developed before the age of one month but they were not present in all individuals before the age of 3 or 4 months. The centres for the proximal epiphysis of the fibula, and for the patella, first make their appearance between $2\,^1/_2$ and 3 years of age and are not present in all boys before the age of 5 years.

In the ossification area of the hip-joint, the proximal epiphysis of the femur appears before one year of age while the greater trochanter begins to ossify at 2—3 years of age and the lesser trochanter was not found in any case before the age of 5.

It would seem logical to expect that the ossific centres for the foot would develop earlier than those for the hand, since in man greater demands are sooner placed on the stability of the foot than on that of the hand. This was not the case, however, at earlier stages in the development of the species. Obviously, there has been no adjustment to fit the erect position in man, since no noticeable difference between the development of hand and foot could be distinguished. The chief point of interest is to see whether homologous bones in both extremities developed their ossific centres at the same time. The difference in the medians for homologous bones in both extremities, in boys and girls is shown in tables 7 and 8.

It will be seen that the lower limbs develop their centres somewhat later than the upper limbs. The difference here is statistically significant both for boys and for girls. The differences between the limbs are by no means inconsiderable. When calculated in percent of the median the difference between the medians amounts to one-third of the median. If we compare boys and girls we find that the differences between legs and arms seem to be somewhat smaller in girls. When calculated in months, however, the difference is 0.97 ± 2.18. It is consequently smaller than its standard error. If we calculate differences in percent of the respective medians we find that the difference between the sexes is $6.32 \pm 10.1\,\%$; this value is not significant either.

If we compare boys and girls we find that throughout, the girls show earlier ossification and seem to have a smaller

Table 7. Median differences for homologous ossific centres in the two extremities in boys and girls.

Number of ossific centres	Boys		Girls	
	Median difference in months	In per cent of the median of the upper extremity	Median difference in months	In per cent of the median of the upper extremity
10—38	− 0.5	1.6	− 0.2	1.0
11—39	−16.1	70.3	−12.8	97.7
12—40	−16.2	69.5	−13.2	83.0
13—41	−22.6	90.8	−14.8	86.0
14—42	−22.1	84.7	−18.0	105.9
15—43	+ 2.6	8.3	+ 0.7	3.4
16—44	− 4.8	28.1	− 3.8	34.2
17—45	− 6.8	48.6	− 1.3	11.8
18—46	− 5.0	29.8	− 2.0	17.7
19—47	− 5.3	22.6	− 6.5	44.8
20—48	− 0.1	0.4	− 0.9	5.6
21—49	− 2.2	9.5	− 2.6	17.4
22—50	− 3.3	14.4	− 7.5	50.3
23—51	− 0.3	0.9	− 3.7	16.7
24—52	+ 2.3	14.1	+ 0.8	7.3
25—53	− 5.1	14.8	− 6.2	27.1
26—54	−13.3	47.2	− 9.0	44.8
27—55	−12.4	42.3	− 9.2	45.3
28—56	− 4.7	13.5	− 7.2	31.4

Table 8. The average median difference for homologous ossific centres in the two extremities in boys and girls.

$M \pm \varepsilon(M)$ = mean ± standard error of the mean, σ = standard deviation.

	Boys			Girls		
	Number	$M \pm \varepsilon(M)$	σ	Number	$M \pm \varepsilon(M)$	σ
Median difference in months	19	-7.2 ± 1.8	7.7	19	-6.2 ± 1.3	5.5
Median difference in per cent of the upper extremities	19	32.2 ± 6.6	28.9	19	38.5 ± 7.6	32.9

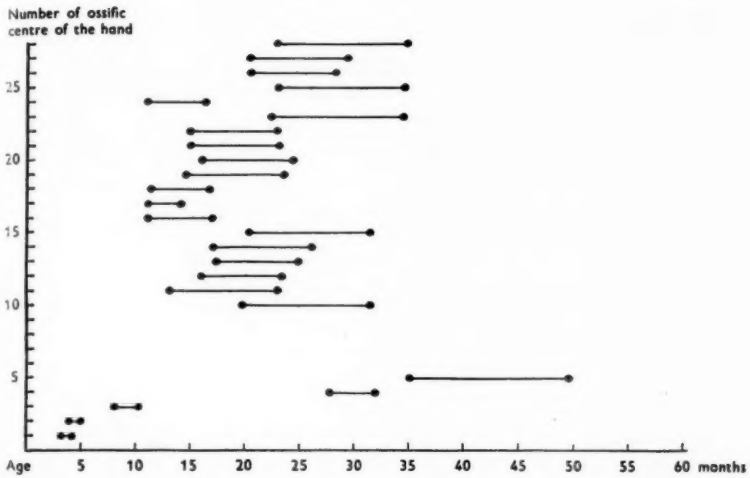


Fig. 12. The differences between the medians for girls and boys in months. The medians of boys and girls are marked with points. The distance between the medians is shown by a thin line, if girls come before boys.

variability. In the case of the individual ossific centres the possibility that the latter feature is due to chance cannot be excluded. As will be seen in tables 5 a, 5 b, and 5 c which also shows the difference between the medians, this difference is always in favour of the girls, with the exception of nos. 57, 58, and 68. The reason why there are no differences for these centres can probably be put down to chance.

These observations indicate that girls develop more rapidly. I have not found any statements in the literature as to whether this is also a feature of the development during embryonic life also, but this does not seem likely, since the differentiation of the ossific centres at birth seems on the whole to be uniform for both boys and girls. The lead held by the girls is also very small during the first months of life, but it increases after the first year. As was shown above, however, such differences between the sexes are present throughout, for all centres except nos. 57, 58, and 68. As will be seen from figures 12—14, the difference varies,

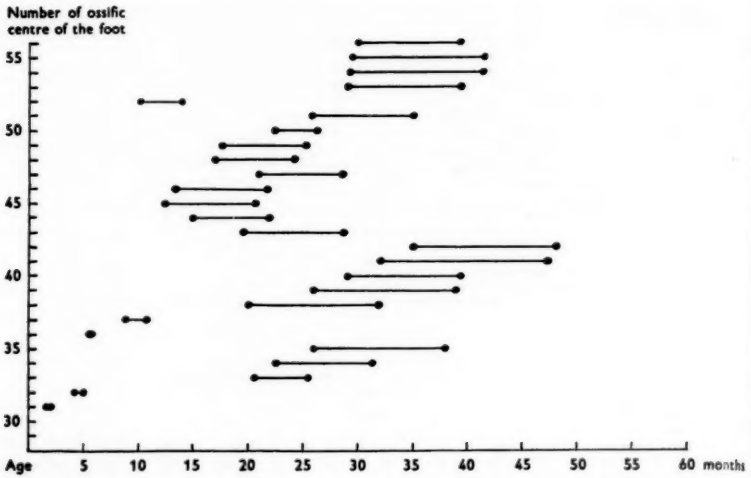


Fig. 13. The differences between the medians for girls and boys in months. The medians of boys and girls are marked with points. The distance between the medians is shown by a thin line, if girls come before boys.

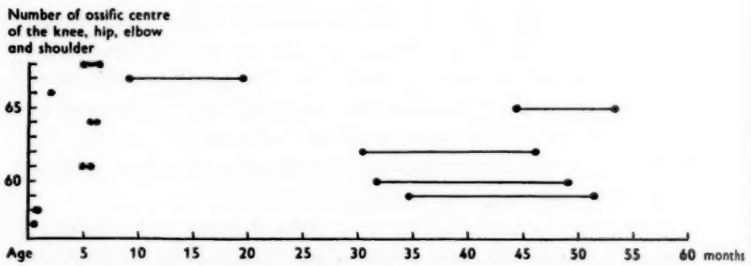


Fig. 14. The differences between the medians for girls and boys in months. The medians of boys and girls are marked with points. The distance between the medians is shown by a thin line, if girls come before boys; the opposite case is marked with a thick line.

a fact which may possibly be ascribed to chance. The tendency is clear, however.

In order to obtain an idea of the variability, the figures have been assembled in another way in table 9.

Table 9. The semi-interquartile range in relation to the median.

Q_1 = first quartile, Q_2 = median, Q_3 = third quartile.

Number of ossific centre	$\frac{Q_3 - Q_1}{2} / Q_2$		Number of ossific centre	$\frac{Q_3 - Q_1}{2} / Q_2$	
	Boys	Girls		Boys	Girls
1	0.15	0 45	35	0.26	0.24
2	0.30	0.33	36	0.25	0.17
3	0.26	0.28	37	0.59	0.19
4	0.38	0.22	38	0.09	0.11
5	0.21	0.29	39	0.13	0.11
6	—	—	40	0.15	0.11
7	—	—	41	0.09	0.12
8	—	—	42	0.12	0.16
9	—	—	43	0.15	0.07
10	0.14	0.17	44	0.20	0.26
11	0.17	0.27	45	0.21	0.24
12	0.20	0.18	46	0.15	0.31
13	0.15	0.17	47	0.16	0.12
14	0.13	0.18	48	0.14	0.27
15	0.11	0.15	49	0.27	0.35
16	0.27	0.19	50	0.33	0.31
17	0.16	0.16	51	0.26	0.30
18	0.32	0.20	52	0.24	0.20
19	0.17	0.18	53	0.17	0.18
20	0.18	0.22	54	0.14	0.25
21	0.19	0.28	55	0.12	0.22
22	0.19	0.24	56	0.16	0.21
23	0.19	0.16	57	0.50	0.50
24	0.33	0.40	58	1.00	1.19
25	0.16	0.17	59	0.15	0.19
26	0.16	0.26	60	0.13	0.09
27	0 17	0.25	61	0.25	0.27
28	0.18	0.14	62	0.09	0.11
29	—	—	63	—	—
30	—	—	64	0.42	0.32
31	0.73	0.56	65	—	0.15
32	0.33	0.31	66	0.69	0.74
33	0.38	0.31	67	0.35	0.16
34	0.20	0.12	68	0 61	0.13

It must, however, be expected that the variability bears a certain relation to the median. If the median is low there is less room for variability. To allow for this, we have placed the mean quartile in relation to the median for the boys and the girls. It was found that among 68 ossific centres the girls had a greater variability in 35 cases and the boys in 23 cases. The relation could not be calculated for 3 centres. On the whole there was no difference between girls and boys with respect to the variability. The difference is 0.004 ± 0.03. It can therefore be considered to be due to chance.

CHAPTER 4.

Comparison between the Skeletal Development in the two Halves of the Body.

Considering the great variability existing normally with respect to each ossific centre, it is possible, or even fairly probable, that the centres do not develop at a uniform rate in both halves of the body. This must be especially the case with centres having the greatest range of variability. In order to ascertain whether there actually is a difference in development in the right and left halves of the body, and if so to what degree, the bones of the extremities on both sides were roentgenographed simultaneously in a material consisting of 59 children, 28 boys and 31 girls of different ages. The result is shown in tables 10 and 11.

According to tables 10 and 11 the ossific centres causing deviation between right and left side are found in the hand and foot. Exceptions are the greater trochanter (No. 62) and the proximal epiphysis of the radius (No. 65).

It will be seen from these tables that there is a difference between the skeletal development in the two halves, although this difference is small. The development runs parallel until the end of the first year, when the phalangeal epiphyses begin to appear, this taking place earlier in girls than in boys. There is, as was mentioned before, great variability as regards the appearance and development of the phalangeal centres, and it seems likely, therefore, that if there were any differences between the two halves they would, if anywhere, be found just among the phalanges. And with few exceptions this actually proves to be the case. It will be observed that it is only with a few isolated centres that these divergences occur, and only on two occasions was it found that two ossific centres were included in the difference.

Table 10. Ossification in the two halves of the body in boys.

Age (months)	No. of ossific centres		No. of centre causing deviation
	Right side	Left side	
5	8	8	—
5	13	13	—
6	13	13	—
7	11	11	—
10	13	13	—
10	14	14	—
10	15	15	—
10	23	24	51
11	26	26	—
15	34	34	—
15	23	23	—
18	43	41	21, 43
24	38	37	10
27	38	39	10
30	45	44	10
33	51	50	15
36	54	53	40
39	49	50	33
39	36	37	14
42	63	63	—
43	46	47	21
45	48	48	—
48	61	62	65
54	56	56	—
57	55	54	62
57	61	61	—
60	64	64	—
60	67	67	—

Table 11. Ossification in the two halves of the body in girls.

Age (months)	No. of ossific centres		No. of centre causing deviation
	Right side	Left side	
4	13	13	—
4	12	12	—
6	12	12	—
6	14	14	—
6	15	15	—
6	13	13	—
9	16	15	32
10	16	17	52
10	23	22	48
11	31	32	20
15	44	45	50
17	16	16	—
18	45	47	15, 35
18	46	47	53
21	58	58	—
27	45	45	—
27	54	54	—
30	63	63	—
30	50	50	—
36	56	55	40
36	60	61	42
36	60	60	—
37	56	57	42
39	61	62	5
42	58	58	—
45	63	63	—
45	63	63	—
48	66	66	—
48	61	61	—
48	62	63	6
54	60	60	—

An obvious explanation to account for this difference in skeletal development would seem to be that there may be a more rapid differentiation of the half of the body which is possibly

ahead in development, the right half in right-handed children and the left in left-handed ones. This does not seem to be the case in the material examined, however. All the 59 children were right-handed (or at least all those old enough to be judged from this standpoint). As can be seen from the tables, there is no difference in the number of deviations in the right or left half of the body in the boys; in 9 of the girls, the left side even had several ossific centres as against only three on the right side.

CHAPTER 5.

Correlation Calculations.

It is obvious that there is a correlation between the individual ossific centres as regards their dates of appearance, and also that the appearance of the centres is correlated with the growth. If the differentiation proceeded absolutely parallel in different tissues and different parts of the body we would have an absolute connection, and from the development of one single ossific centre we could arrive at the development of the others. But the single centre varies. In one individual it appears earlier, in another later. This is due to a number of different factors. First of all, it depends on the age of the child when it was born, i. e. on whether the child was born more or less at full term, and also on the length of the mother's pregnancy.

Information as to the duration of the pregnancy was not available in this material. But the figures for the weight and height were obtained. Both figures vary, as was mentioned in chapter 2. This is due, among other factors, to the fact that the length of the pregnancy had varied, and is without doubt a reflection of the fact that the children were at different stages of development at birth. The date of appearance of the ossific centres ought, consequently, also to vary. Another cause is, of course, the diet and other factors of environment and a third, hereditary factors influencing the rate of development.

Because of this variability the ossific centres ought not, of course, to be absolutely correlated with one another with respect to times of appearance. In order to examine the conditions existing, a few centres which in respect of median and quartiles more or less coincide have been selected for study (table 12).

Table 12. Absolute and percentual distribution of certain ossific centres with a comparable developmental phase.

Age, months	Total number of observations = N	All cases showing one or both of the centres		Cases showing only the first-mentioned ossific centre		Cases showing only the last-mentioned ossific centre		Cases showing both of the mentioned ossific centres	
		Number = n	In per cent of N	Number	In per cent of n	Number	In per cent of n	Number	In per cent of n
Ossific centres 4 and 5									
0—21	378	28	7.4	19	67.9	0	0	9	32.1
0—33	461	59	12.8	38	64.4	0	0	21	35.6
0—48	525	105	20.0	60	57.1	0	0	45	42.9
0—54	545	125	22.9	66	52.8	0	0	59	47.2
Ossific centres 12 and 13									
0—18	363	11	3.0	2	18.2	0	0	9	81.8
0—24	401	30	7.5	7	23.3	0	0	23	76.7
0—27	423	48	11.3	11	22.9	0	0	37	77.1
0—36	476	97	20.4	13	13.4	0	0	84	86.6
Ossific centres 26 and 27									
0—24	401	19	4.7	2	10.5	0	0	17	89.5
0—27	423	29	6.9	5	17.2	1	3.4	23	79.3
0—30	439	40	9.1	7	17.5	1	2.5	32	80.0
0—54	545	140	25.7	10	7.1	1	0.7	129	92.2
Ossific centres 10 and 28									
0—27	423	20	4.7	12	60.0	5	25.0	3	15.0
0—30	439	26	5.9	16	61.5	5	19.2	5	19.3
0—33	461	42	9.1	24	57.1	5	11.9	13	31.0
0—52	535	112	20.9	35	31.3	7	6.2	70	62.5

It will be seen from the table that as regards the first pair of ossific centres compared, nos. 4 and 5, no. 4 is present in 7.4 per cent of the material up to the 21st month. At the same time no. 5 is not present in any case in which no. 4 is not present, this being an expression of the fact that they are correlated. On the other hand, no. 5 is present in 32.1 per cent of the cases where no. 4 is present. When we examine the state of affairs in the 33rd month

it is found that centre no. 4 is present in 13 per cent. In 35 per cent of these, centre no. 5 is also present, but no. 5 is still not present in any case in which no. 4 is not present. The figures in the table relative to centres nos. 12 and 13 also show this relation throughout. Only the one centre, that appearing earliest, occurs on its own; not the other which appears later.

If we glance at the table, we find that centre no. 4 comes before no. 5, and centre no. 12 before no. 13. Looking at centres nos. 26 and 27, we find a small difference between median and quartiles in both centres. They can therefore be present independently of one another in a few cases. As can be seen from the table they usually appear together, however. The table is only intended to demonstrate that, as was to be expected, the individual ossific centres are correlated as regards their development, and I shall therefore not attempt to go into this correlation in more detail by means of special calculations of correlation coefficients.

As it can therefore be taken for granted that there is no absolute correlation between the dates of appearance of the different ossific centres, a more complete idea of the situation ought to be obtained by examining different centres simultaneously and indicating how many centres are present at different ages. There are obviously more centres when the child is a little older than there are at an earlier stage. The correlation with the age is illustrated by calculating correlation coefficients in the usual way. In table 13, we find correlation coefficients with age, with height and weight, and with Rohrer's index.

The table shows that there is a strongly positive correlation between age and number of ossific centres. It is 0.96 in boys and 0.93 in girls. We also find an equally strong correlation between height and number of centres. This is mainly due to the fact that there is a correlation between height and age. All these correlations are of the same magnitude and show no essential divergences between boys and girls. We also get a correlation between body weight and ossific centres, but on the other hand there is a strongly negative correlation between Rohrer's index and the number of ossific centres. The smaller the index the

Table 13. Correlations between height, age, weight, Rohrer's index and number of ossific centres.

$r \pm \varepsilon(r)$ = correlation coefficient ± standard error.

Correlation between:	Boys		Girls	
	Number	$r \pm \varepsilon(r)$	Number	$r \pm \varepsilon(r)$
Number of ossific centres and age	569	$+0.96 \pm 0.0033$	563	$+0.93 \pm 0.0054$
Height and number of ossific centres	568	$+0.95 \pm 0.0041$	563	$+0.95 \pm 0.0039$
Height and age	568	$+0.96 \pm 0.0033$	563	$+0.94 \pm 0.0052$
Number of ossific centres and weight	568	$+0.92 \pm 0.0061$	563	$+0.93 \pm 0.0059$
Number of ossific centres and Rohrer's index	568	-0.83 ± 0.013	563	-0.81 ± 0.015

larger the height in relation to weight, and the older the child. However, as here also there seems to be no difference between the correlations, we can place the boys and girls together in order to obtain a larger material.

It is of interest to study the correlation at different ages. It is obvious that in the case of newborn infants and children in the early years of childhood, the height will possibly be a better indicator of the degree of maturity than the age and weight, and will therefore be more strongly correlated with the ossific centres. When the children are older, on the other hand, they will show greater variations in height at one and the same age. It is then probable that the height variation will not only be an expression of the degree of maturity but will also depend on inherited characteristics and other factors having nothing to do with the degree of maturity. The correlation with the height would then be expected to decrease. In order to ascertain whether this is actually what occurs, partial correlations for different age groups have been calculated in table 14.[1]

A partial correlation with the height means that the age is assumed to be constant, and conversely, a partial correlation with the age means that children of the same height but of different

[1] YULE's formulae have been used in the calculation of partial correlations.

Table 14. Correlations and partial correlations between height, age and number of ossific centres.

$r \pm \varepsilon(r)$ = correlation coefficient ± standard error.

Correlation between:	Age, years					
	≤ 1		≤ 2		2—5	
	Number	$r \pm \varepsilon(r)$	Number	$r \pm \varepsilon(r)$	Number	$r \pm \varepsilon(r)$
Correlation						
Height and number of ossific centres	604	$+0.85 \pm 0.011$	791	$+0.84 \pm 0.010$	340	$+0.69 \pm 0.029$
Height and age	604	$+0.90 \pm 0.0075$	791	$+0.91 \pm 0.0061$	340	$+0.84 \pm 0.016$
Number of ossific centres and age	604	$+0.82 \pm 0.013$	791	$+0.86 \pm 0.0086$	340	$+0.65 \pm 0.031$
Partial correlation						
Height and number of ossific centres, age being constant	604	+0.44	791	+0.28	340	+0.34
Age and number of ossific centres, height being constant	604	+0.24	791	+0.44	340	+0.19

ages are compared. From the figures in the table it will be seen that with respect to the correlation coefficients there are no appreciable differences for the different age groups. On the other hand, the partial correlations show that the connection with the height is stronger than the connection with the age, for children under one year, a feature which, from the above-mentioned standpoint, was to be expected.

If we take children up to the age of 2, we find a stronger partial correlation between age and ossific centres than between height and ossific centres. An unexpected circumstance is, however, that for children of between 2 and 5 years of age the partial correlation with height is once again stronger than the partial correlation with age.

CHAPTER 6.

Normal Tables for the Development of the Ossific Centres.

The practical interest of this investigation is attached to the construction of normal tables for the dates of appearance of the ossific centres. With the aid of these tables it is possible to judge whether children display abnormal deviations from the normal standards.

In the preceding chapter it was shown that the individual centres appear at very varying dates. The range of variation seems to be different for different centres, but the fact that it is large for some centres and small for others may be due to chance.

The degree of maturity will obviously be judged with a greater measure of certainty if the judgment is based on several ossific centres than if only one centre is used. How great the degree of certainty will be will depend on the correlation between the ossific centres. The question of the connection between the centres has been discussed in a preceding chapter.

In tables 15—18, the mean and the standard deviation have been calculated for the number of ossific centres with increasing age and height. These tables can be used for the examination of Swedish children in clinical practice.

In tables 19—22 we find the mean and σ, $2\,^1/_2\,\sigma$ and $3\,\sigma$ on each side of the mean. The probability of a deviation at a distance of $1\,\sigma$ from the mean is $\frac{1}{3{,}15}$. Deviations at greater distances than $3\,\sigma$ can be regarded as definitely pathologic and have a probability of less than $\frac{1}{368}$. Deviations at greater distances

Table 15. Number of ossific centres at different ages in boys and girls.

$M \pm \varepsilon(M)$ = mean ± standard error of the mean. σ = standard deviation.

Age, months	Boys			Girls		
	Number	$M \pm \varepsilon(M)$	σ	Number	$M \pm \varepsilon(M)$	σ
1	45	4.8 ± 0.28	1.9	48	4.7 ± 0.28	1.9
2	37	5.7 ± 0.33	2.0	37	6.2 ± 0.38	2.3
3	32	6.5 ± 0.35	2.0	25	7.6 ± 0.51	2.5
4	24	8.9 ± 0.56	2.8	21	8.5 ± 0.60	2.8
5	28	9.8 ± 0.45	2.4	19	10.4 ± 0.45	2.0
6	26	11.2 ± 0.46	2.4	22	11.5 ± 0.36	1.7
7	21	12.5 ± 0.63	2.9	17	12.9 ± 0.34	1.4
8	22	13.0 ± 0.37	1.7	22	14.6 ± 0.74	3.5
9	20	13.6 ± 0.61	2.7	23	16.3 ± 0.50	2.4
10	19	15.2 ± 0.80	3.5	22	18.1 ± 0.93	4.4
11	16	15.8 ± 0.81	3.2	20	22.7 ± 1.54	6.9
12	19	16.5 ± 1.14	4.9	19	25.1 ± 2.00	8.7
13—15	26	19.9 ± 1.24	6.3	28	28.6 ± 1.74	9.2
16—18	28	23.5 ± 1.59	8.4	26	32.9 ± 1.73	8.8
19—21	15	25.5 ± 2.17	8.4	20	41.3 ± 1.92	8.6
22—24	23	32.3 ± 1.92	9.2	21	47.2 ± 1.55	7.1
25—27	22	36.8 ± 1.18	5.5	19	50.8 ± 1.11	4.8
28—30	16	39.8 ± 2.09	8.4	23	53.2 ± 1.35	6.5
31—33	22	44.1 ± 1.01	4.8	18	55.8 ± 1.13	4.8
34—36	15	48.5 ± 1.49	5.8	18	60.5 ± 0.70	3.0
37—42	21	49.5 ± 1.50	6.9	31	59.5 ± 0.81	4.5
43—48	27	56.6 ± 0.95	4.9	24	61.4 ± 1.34	6.6
49—54	20	59.3 ± 1.24	5.5	19	63.5 ± 0.50	2.2
55—60	24	61.8 ± 0.71	3.5	21	64.2 ± 0.51	2.3

than $2\,^1/_2\,\sigma$ can also be regarded as probably pathologic, although it sometimes happens that normal individuals with this deviation are encountered. The probability of this deviation is $\frac{1}{80{,}6}$ at the lowest.

It must be stressed at the outset, however, that the value of such tables is rather limited. It has been found that the rate of development in respect of height has increased during the past fifty years.

Table 16. Number of ossific centres at different heights in boys and girls.

$M \pm \varepsilon(M)$ = mean ± standard error of the mean. σ = standard deviation.

Height, cm	Boys			Girls		
	Number	$M \pm \varepsilon(M)$	σ	Number	$M \pm \varepsilon(M)$	σ
<50	25	3.3 ± 0.23	1.1	44	3.7 ± 0.19	1.3
50— 55	48	5.4 ± 0.16	1.1	41	6.4 ± 0.27	1.7
55— 60	39	6.7 ± 0.27	1.7	43	8.3 ± 0.29	1.9
60— 65	58	9.1 ± 0.27	2.1	42	11.2 ± 0.23	1.5
65— 70	54	12.1 ± 0.25	1.8	44	13.6 ± 0.30	2.0
70— 75	78	14.5 ± 0.31	2.7	64	18.6 ± 0.56	4.5
75— 80	38	19.8 ± 0.82	5.1	60	28.5 ± 0.99	7.7
80— 85	43	25.0 ± 1.04	6.8	32	41.3 ± 1.63	9.2
85— 90	35	34.8 ± 1.23	7.3	41	48.1 ± 1.16	7.4
90— 95	36	43.2 ± 0.97	5.8	38	54.1 ± 0.90	5.6
95—100	27	45.1 ± 1.52	7.9	38	59.3 ± 0.57	3.5
100—105	35	54.1 ± 0.80	4.7	33	61.4 ± 0.53	3.1
105—110	28	59.3 ± 0.88	4.7	21	64.0 ± 0.44	2.0
110—115	13	60.5 ± 1.37	4.9	17	64.9 ± 0.55	2.3

Children of 15 years of age are 15 cm taller than they were fifty years ago. In full-grown persons the difference is only 5 cm. (Cf. BROMAN, DAHLBERG, LICHTENSTEIN, 1942.) These authors state, with reference to a theory propounded by DAHLBERG in 1931, that this increased rate of growth is due to environmental factors. A better environment causes a more rapid rate of growth, but the end-result is not materially altered by the environment. The rate at which this result is achieved increases, however.

Analogously, it can be expected that when the environment is improved the rate at which the ossific centres develop will increase. This rate thus ought to differ among people living in different social environments. In proportion as the differences disappear and optimal conditions are reached we can expect to obtain a curve relative to the development of the ossific centres which will be a permanently usable one. It is not possible to decide how close we have come to this goal.

Table 17. A study of the normal development of the ossific centr

$M \pm \sigma$ = mean ± standard deviation.

Age, months	Boys		
	Number	$M \pm \sigma$	Ossific centres which assume the mean
1	45	4.8±1.9	29.30.57.58.(1.31.66.)
2	37	5.7±2.0	29.30.31.57.58.66.(1.68.)
3	32	6.5±2.0	29-31.57.58.66.68.(1.64.)
4	24	8.9±2.8	1.2.29-31.57.58.66.68.(32.36.61.)
5	28	9.8±2.4	1.2.29-32.57.58.66.68.(36.61.)
6	26	11.2±2.4	1.2.29-32.36.57.58.66.68.(61.64.)
7	21	12.5±2.9	1.2.29-32.36.57.58.61.64.66.68.(3.37.67.)
8	22	13.0±1.7	1.2.29-32.36.57.58.61.64.66.68.(3.37.)
9	20	13.6±2.7	1-3.29-32.36.57.58.61.64.66.68.(17.37.67.)
10	19	15.2±3.5	1-3.29-32.36.37.57.58.61.64.66.68.(16-18.67.)
11	16	15.8±3.2	1-3.29-32.36.37.57.58.61.64.66-68.(16-18.)
12	19	16.5±4.9	1-3.17.29-32.36.37.57.58.61.64.66-68.(16.18.33.45.49.)
13—15	26	19.9±6.3	1-3.16.17.24.29-32.36.37.52.57.58.61.64.66-68.(11.18.45.49.33.50.)
16—18	28	23.5±8.4	1-3.16-18.24.29-32,36.37.44-46.52.57.58.61.64.66-68.(4.11-13.33.48-50.)
19—21	15	25.5±8.4	1-3.11.16-18. 24. 29-32. 36. 37. 44-46. 49. 52.57.58.61.64.66-68.(4.12.13.21.22.33. 49.50.)
22—24	23	32.3±9.2	1-3.11-13.16-22.24.29-33.36.37.44-46.48.49.52.57.58.61.64.66-68(4.14.23.26.27. 34.43.47.50.)
25—27	22	36 8±5.5	1-4.11-14.16-22.24.29-33. 36. 37.43-46.48-50.52.57.58.61.64.66-68.(10.15.26.27. 34.47.)
28—30	16	39.8±8.4	1-4.11-14.16-22.24.26.29-33.36.37.43-50. 52. 57. 58.61.64.66-68.(10.15.23.25.27. 34.38.51.)
31—33	22	44.1±4.8	1-4.10-22.24.26.27.29-34.36-38.43-50.52.57.58.61.64.66-68.(23.25.28.35.51.)
34—36	15	48.5±5.8	1-4.10-27.29-34.36-38.43-52.57.58.61.64.66-68.(5.28.35.39.53.54.)
37—42	21	49.5±6.9	1-4.10-34.36-38.43-52.57.58.61.64.66-68.(5.35.39.40.53-55.)
43—48	27	56.6±4.9	1-5.10-40.43-58.61.64.66-68.(41.42.59.60.62.)
49—54	20	59.3±5.5	1-5.10-41.43-58.61-62.64.66-68.(6.7.42.59.60.65.)
55—60	24	61.8±3.5	1-6.10-62.64.66-68.(7-9.65.)

centres during infancy and childhood in boys and girls.

(Centres in brackets may also be present.)

Age, months	Girls		
	Number	$M \pm \sigma$	Ossific centres which assume the mean
1	48	4.7±1.9	29-31.57.58.(1.66.)
2	37	6.2±2.3	29-31.57.58.66.(1.68.)
3	25	7.6±2.5	1.29-32.57.58.66.(2.36.68.)
4	21	8.5±2.8	1.2.29-32.57.58.66.(36.61.64.)
5	19	10.4±2.0	1.2.29-32.57.58.61.66.(36.64.)
6	22	11.5±1.7	1.2.29-32.36.57.58.61.64.66.(37.68.)
7	17	12.9±1.4	1.2.29-32.36.57.58.61.64.66.68.(37.)
8	22	14.6±3.5	1-3.29-32.36.37.57.58.61.64.66.68.(24.52.67.)
9	23	16.3±2.4	1-3.29-32.36.37.57.58.61.64.66-68.(24.52.)
10	22	18.1±4.4	1-3.24.29-32.36.37.52.57.58.61.64.66-68.(16-18.33.)
11	20	22.7±6.9	1-3.16-18.24.29-32.36.37.45.46.52.57.58.61.64.66-68.(11.12.21.33.44.48.49.)
12	19	25.1±8.7	1-3.16-18.24.29-32.36.37.45.46. 48. 49. 52. 57.58.61.64.66-68.(11.12.20-22.26.33. 44.50.)
13—15	28	28.6±9.2	1-3.11.16-19.21.22.24.29-32.36. 37. 45. 46. 48.49.52.57.58.61.64.66-68.(12.13.20. 23.26.27.33.44.50.)
16—18	26	32.9±8.8	1-3.11-13.16-22.24.29-32.36.37. 44-46. 48. 49.52.57.58.61.64.66-68.(10.14.15.23. 26.27.33.38.50.)
19—21	20	41.3±8.6	1-3.10-22.24.26.27. 29-33. 36-38. 43-46. 48. 49. 52. 57.58.61.64.66-68.(4.23.25.28. 34.47.50.51.54.)
22—24	21	47.2±7.1	1-3.10-34.36-38.43-50.52.57.58.61.64.66-68.(4.35.51.53-56.)
25—27	19	50.8±4.8	1-4.10-39.43-52.57.58.61.64.66-68.(40.53-56.)
28—30	23	53.2±6.5	1-4.10-40.43-53.57.58.61.64.66-68.(5.41.54-56.60.62.)
31—33	18	55.8±4.8	1-4.10-40.43-54.57.58.60-62.64.66-68.(5.41.55.56.59.)
34—36	18	60.5±3.0	1-4.10-58.60-62.64.66-68.(5.59.65.)
37—42	31	59.5±4.5	1-4.10-58.60-62.64.66-68.(5-7.59.65.)
43—48	24	61.4±6.6	1-4.10-62.64.66-68.(5-8.65.)
49—54	19	63.5±2.2	1-5.10-62.64-68.(6-8.)
55—60	21	64.2±2.3	1-7.10-62.64-68.(8.9.)

Table 18. A study of the normal development of the ossific

$M \pm \sigma$ = mean ± standard deviation.

Height, cm.	Boys		
	Number	$M \pm \sigma$	Ossific centres which assume the mean
<50	25	3.3±1.1	29.30.57.(58.)
50—55	48	5.4±1.1	29-31.57.58.(66.)
55—60	39	6.7±1.7	29-31.57.58.66.(1.68.)
60—65	58	9.1±2.1	1.2.29-31.36.57.58.66.(32.68.)
65—70	54	12.1±1.8	1.2.29-32.36.57.58.61.66.68.(3.64.)
70—75	78	14.5±2.7	1-3.29-32.36.57.58.61.64.66.68.(4.37.67.)
75—80	38	19.8±5.1	1-3.16-18.24.29-32.36.37.52.57.58.61.64.66.68.(4.19.45.49.67.)
80—85	43	25.0±6.8	1-3.11.16-18.22.24.29-32.36.37.44.45.52.57.58.61.64.66-68.(12.13.19-21.33.46.)
85—90	35	34.8±7.3	1-3.11-14.16-22.24.26.29-32.36.37.44-46.48.49.52.57.58.61.64.66-68.(4.10.27.33.34.47.50.)
90—95	36	43.2±5.8	1-3.10-22.24.26.27.29-34.36.37.43-50.52.57.58.61.64.66-68.(4.25.38.51.53.54.
95—100	27	45.1±7.9	1-4.10-22.24.26.27.29-34.36-38.43-50.52.57.58.61.64.66-68.(23.25.28.35.39.51.53.54.)
100—105	35	54.1±4.7	1-4.10-39.43-55.57.58.61.64.66-68.(40-42.56.62.)
105—110	28	59.3±4.7	1-4.10-41.43-59.61.62.64.66-68.(5.6.42.60.65.)
110—115	13	60.5±4.9	1-5.10-59.61.62.64.66-68.(6.7.8.60.65.)

Another reservation that must be made is that the material is not very large. Without doubt, however, it is the largest hitherto described, and embraces many areas of ossification and a relatively large number of ossific centres. Viewed from the statistical standpoint, it would perhaps have been still better if the material had been larger. Nevertheless, it can be said to be sufficiently large to give a correct idea of the development.

The figures shown in tables 19—22 have been represented graphically in figures 15—18.

According to these figures, the increase in the number of ossific centres follows a slightly convex curve for the boys. Among the girls the convexity is more pronounced. The curves show the development up to the age of 5 years and to the height of 115 cm, by which age 67 ossific centres have appeared. In the higher ages there are not many more centres than this in the parts of the

centres in children of different heights during infancy and childhood.

(Centres in brackets may be present.)

Height, cm.	Girls		
	Number	$M \pm \sigma$	Ossific centres which assume the mean
<50	44	3.7±1.3	29.30.57.58.(31.)
50—55	41	6.4±1.7	29-31.57.58.66.(1.68.)
55—60	43	8.3±1.9	1.29-32.57.58.66.(2.68.)
60—65	42	11.2±1.5	1.2.29-32.36.57.58.61.66.(64.68.)
65—70	44	13.6±2.0	1.2.29-32.36.57.58.61.64.66.68.(37.67.)
70—75	64	18.6±4.5	1-3.24.29-32.36.37.52.57.58.61.64.66-68.(16-18.46.49.)
75—80	60	28.5±7.7	1-3.11.16-19.21.22.24.29-32.36.37.44-46.52.57.58.61.64.66-68.(12-14.20.23. 26.48.49.)
80—85	32	41.3±9.2	1-3.10-14.16-22.24.26.27.29-33.36-38.43-49.52.57.58.61.64.66-68.(4.15.23. 25.28.34.50.51.54.)
85—90	41	48.1±7.4	1-3.10-34.36-39.43-50.52.57.58.61.64.66-68.(4.35.51.53-56.)
90—95	38	54.1±5.6	1-4.10-40.43-54.57.58.61.64.66-68.(5.41.42.55.56.60.)
95—100	38	59.3±3.5	1-4.10-41.43-58.60-62.64.66-68.(5.42.59.65.)
100—105	33	61.4±3.1	1-4.10-62.64.66-68.(5.6.65.)
105—110	21	64.0±2.0	1-6.10-62.64-68.(7.8.)
110—115	17	64.9±2.3	1-7.10-62.64-68.(8.9.)

skeleton examined here. Possibly a wider view might be gained by examining the parts of the skeleton not included in this investigation, but the gain could not be so very much greater. The author intends, however, to use a method recommended by DAHLBERG, namely to measure the size of the ossific centres by measuring roentgenograms with the aid of a planimeter. Quite a number of interesting features can be expected to come to light with this method.

It has already been shown that differences in the curves will become apparent if boys and girls are compared. The difference between the means is shown in more detail in figures 19 and 20.

Looking first at the differences between boys and girls in the absolute figures for the number of ossific centres, it will be seen that they increase with age and with height after the age of 10 months and after a height of 70 cm. Before the age of 10

Table 19. Number of ossific centres in boys at different months of age.

Age, months	Average number of ossific centres $= M$	Deviation $= \sigma$	Range of variation					
			$M-3\sigma$	$M-2\frac{1}{2}\sigma$	$M-\sigma$	$M+\sigma$	$M+2\frac{1}{2}\sigma$	$M+3\sigma$
1	4.8	1.9	0	0	2.9	6.7	9.6	10.5
2	5.7	2.0	0	0.7	3.7	7.7	10.7	11.7
3	6.5	2.0	0.5	1.5	4.5	8.5	11.5	12.5
4	8.9	2.8	0.5	1.9	6.1	11.7	15.9	17.3
5	9.8	2.4	2.6	3.8	7.4	12.2	15.8	17.0
6	11.2	2.4	4.0	5.2	8.8	13.6	17.2	18.4
7	12.5	2.9	3.8	5.2	9.6	15.4	19.8	21.2
8	13.0	1.7	7.9	8.7	11.3	14.7	17.3	18.1
9	13.6	2.7	5.5	6.8	10.9	16.3	20.4	21.7
10	15.2	3.5	4.7	6.4	11.7	18.7	24.0	25.7
11	15.8	3.2	6.2	7.8	12.6	19.0	23.8	25.4
12	16.5	4.9	1.8	4.2	11.6	21.4	28.8	31.2
13—15	19.9	6.3	1.0	4.1	13.6	26.2	35.7	38.8
16—18	23.5	8.4	0	2.5	15.1	31.9	44.5	48.7
19—21	25.5	8.4	0.3	4.5	17.1	33.9	46.5	50.7
22—24	32.3	9.2	4.7	9.3	23.1	41.5	55.3	59.9
25—27	36.8	5.5	20.3	23.0	31.3	42.3	50.6	52.3
28—30	39.8	8.4	14.6	18.8	31.4	48.2	60.8	65.0
31—33	44.1	4.8	29.7	32.1	39.3	48.9	56.1	58.5
34—36	48.5	5.8	31.1	34.0	42.7	54.3	63.0	65.9
37—42	49.5	6.9	28.8	32.2	42.6	56.4	66.8	70.2
43—48	56.6	4.9	41.9	44.3	51.7	61.5	68.9	71.3
49—54	59.3	5.5	42.8	45.5	53.8	64.8	73.1	75.8
55—60	61.8	3.5	51.3	53.0	58.3	65.3	70.6	72.3

months and before a height of 70 cm they are very small. The differences then remain fairly constant up to the age of $3\frac{1}{2}$—4 years, that is, up to a height of about 105 cm, after which they decrease. If we calculate the differences in a percentage of the boys' values this feature becomes still more evident. (Tables 23 and 24, figs. 21 and 22.)

The percentual difference between boys and girls reaches its maximum at the age of about 20 months and a height of 85 cm,

Table 20. Number of ossific centres in girls at different months of age.

Age, months	Average number of ossific centres $= M$	Deviation $= \sigma$	Range of variation					
			$M-3\sigma$	$M-2\frac{1}{2}\sigma$	$M-\sigma$	$M+\sigma$	$M+2\frac{1}{2}\sigma$	$M+3\sigma$
1	4.7	1.9	0	0	2.8	6.6	9.5	10.4
2	6.2	2.3	0	0.4	3.9	8.5	12.0	13.1
3	7.6	2.5	0.1	1.3	5.1	10.1	13.9	15.1
4	8.5	2.8	0.1	1.5	5.7	11.3	15.5	16.9
5	10.4	2.0	4.4	5.4	8.4	12.4	15.4	16.4
6	11.5	1.7	6.4	7.2	9.8	13.2	15.8	16.6
7	12.9	1.4	8.7	9.4	11.5	14.3	16.4	17.1
8	14.6	3.5	4.1	5.8	11.1	18.1	23.4	25.1
9	16.3	2.4	9.1	10.3	13.9	18.7	22.3	23.5
10	18.1	4.4	4.9	7.1	13.7	22.5	29.1	31.3
11	22.7	6.9	2.0	5.4	15.8	29.6	40.0	43.4
12	25.1	8.7	0	3.3	16.4	33.8	46.9	51.2
13—15	28.6	9.2	1.0	5.6	19.4	37.8	51.6	56.2
16—18	32.9	8.8	6.5	10.9	24.1	41.7	54.9	59.3
19—21	41.3	8.6	15.5	19.8	32.7	49.9	62.8	67.1
22—24	47.2	7.1	25.9	29.4	40.1	54.3	65.0	68.5
25—27	50.8	4.8	36.4	38.8	46.0	55.6	62.8	65.2
28—30	53.2	6.5	33.7	36.9	46.7	59.7	69.5	72.7
31—33	55.8	4.8	41.4	43.8	51.0	60.6	67.8	70.2
34—36	60.5	3.0	51.5	53.0	57.5	63.5	68.0	69.5
37—42	59.5	4.5	46.0	48.2	55.0	64.0	70.8	73.0
43—48	61.4	6.6	41.6	44.9	54.8	68.0	77.9	81.2
49—54	63.5	2.2	56.9	58.0	61.3	65.7	69.0	70.1
55—60	64.2	2.3	57.3	58.4	61.9	66.5	70.0	71.1

when it amounts to 60—65 per cent. Towards the age of 5 years the differences are evened up.

It will be seen that both age and height can be used in the estimation of ossific centre development. In the earlier years, however, the height is better then the age, as the correlation calculations in a preceding chapter have shown; in older children both methods can be used with equally good results.

Table 21. Number of ossific centres in boys at different heights.

Height, cm	Average number of ossific centres = M	Deviation = σ	Range of variation					
			$M-3\sigma$	$M-2\,1/2\,\sigma$	$M-\sigma$	$M+\sigma$	$M+2\,1/2\,\sigma$	$M+3\sigma$
<50	3.3	1.1	0	0.5	2.2	4.4	6.1	6.6
50— 55	5.4	1.1	2.1	2.6	4.3	6.5	8.2	8.7
55— 60	6.7	1.7	1.6	2.4	5.0	8.4	11.0	11.8
60— 65	9.1	2.1	2.8	3.8	7.0	11.2	14.4	15.4
65— 70	12.1	1.8	6.7	7.6	10.3	13.9	16.6	17.5
70— 75	14.5	2.7	6.4	7.7	11.8	17.2	21.3	22.6
75— 80	19.8	5.1	4.5	7.0	14.7	24.9	32.6	35.1
80— 85	25.0	6.8	4.6	8.0	18.2	31.8	42.0	45.4
85— 90	34.8	7.3	12.9	16.5	27.5	42.1	53.1	56.7
90— 95	43.2	5.8	25.8	28.7	37.4	49.0	57.7	60.6
95—100	45.1	7.9	21.4	25.3	37.2	53.0	64.9	68.8
100—105	54.1	4.7	40.0	42.3	49.4	58.8	65.9	68.2
105—110	59.3	4.7	45.2	47.5	54.6	64.0	71.1	73.4
110—115	60.5	4.9	45.8	48.2	55.6	65.4	72.8	75.2

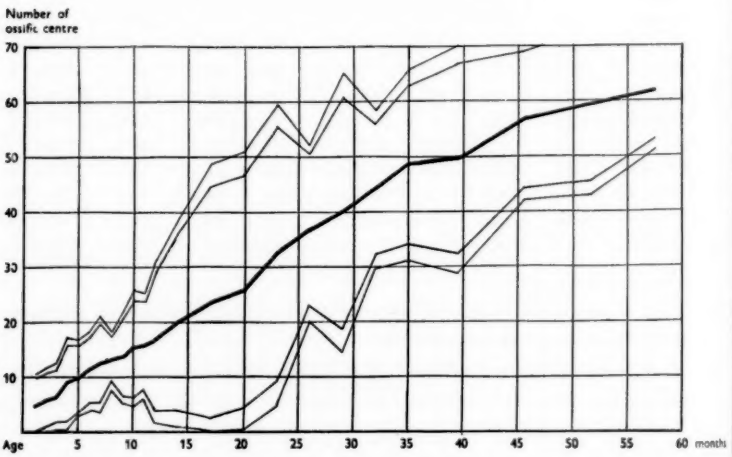


Fig. 15. Number of ossific centres in boys of different ages. Thick line = means. The thin lines delimit the spaces between $2\,1/2\,\sigma$ and $3\,\sigma$ on each side of the means.

Table 22. Number of ossific centres in girls at different heights.

Height, cm	Average number of ossific centres = M	Deviation = σ	Range of variation					
			$M-3\sigma$	$M-2\,1/2\,\sigma$	$M-\sigma$	$M+\sigma$	$M+2\,1/2\,\sigma$	$M+3\sigma$
<50	3.7	1.3	0	0.4	2.4	5.0	7.0	7.6
50— 55	6.4	1.7	1.3	2.1	4.7	8.1	10.7	11.5
55— 60	8.3	1.9	2.6	3.5	6.4	10.2	13.1	14.0
60— 65	11.2	1.5	6.7	7.4	9.7	12.7	15.0	15.7
65— 70	13.6	2.0	7.7	8.6	11.6	15.6	18.6	19.6
70— 75	18.6	4.5	5.1	7.3	14 1	23.1	29.9	32.1
75— 80	28.5	7.7	5.4	9.2	20.8	36.2	47.8	51.6
80— 85	41.3	9.2	13.7	18.3	32.1	50.5	64.3	68.9
85— 90	48.1	7.4	25.9	29.6	40.7	55.5	66.6	70.3
90— 95	54.1	5.6	37.3	40.1	48.5	59.7	68.1	70.9
95—100	59.3	3.5	48.8	50.5	55.8	62.8	68.1	69.8
100—105	61.4	3.1	52.1	53.6	58.3	64.5	69.2	70.7
105—110	64.0	2.0	58.0	59.0	62.0	66.0	69.0	70.0
110—115	64.9	2.3	58 0	59.1	62.6	67.2	70.7	71.8

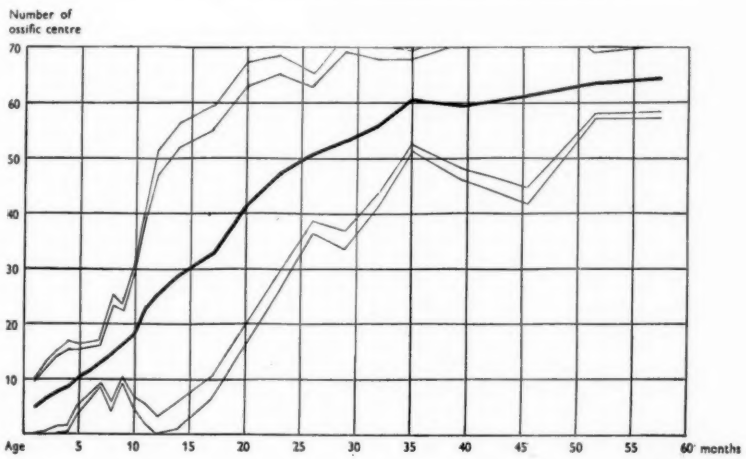


Fig. 16. Number of ossific centres in girls of different ages. Thick line = means. The thin lines delimit the spaces between 2 1/2 σ and 3 σ on each side of the means.

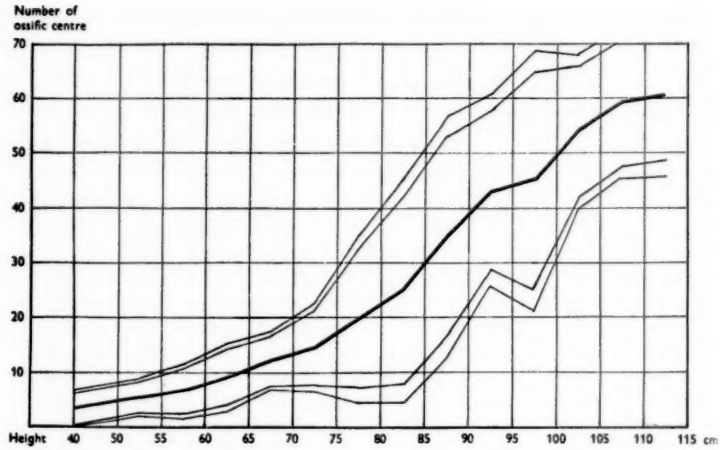


Fig. 17. Number of ossific centres in boys of different heights. Thick line = means. The thin lines delimit the spaces between 2 ½ σ and 3 σ on each side of the means.

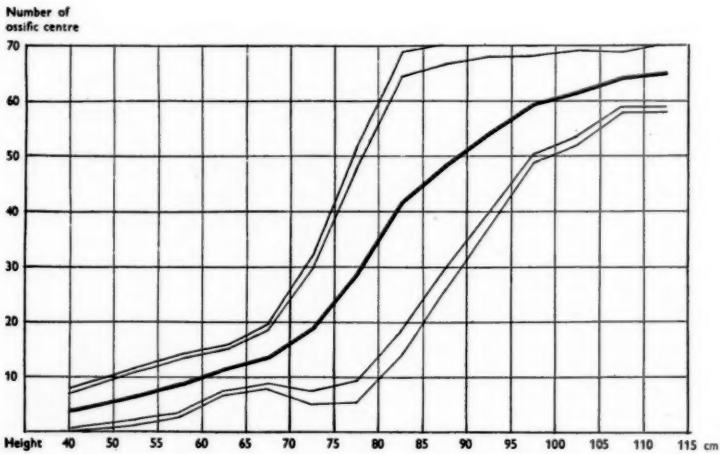


Fig. 18. Number of ossific centres in girls of different heights. Thick lines = means. The thin lines delimit the spaces between 2 ½ σ and 3 σ on each side of the means.

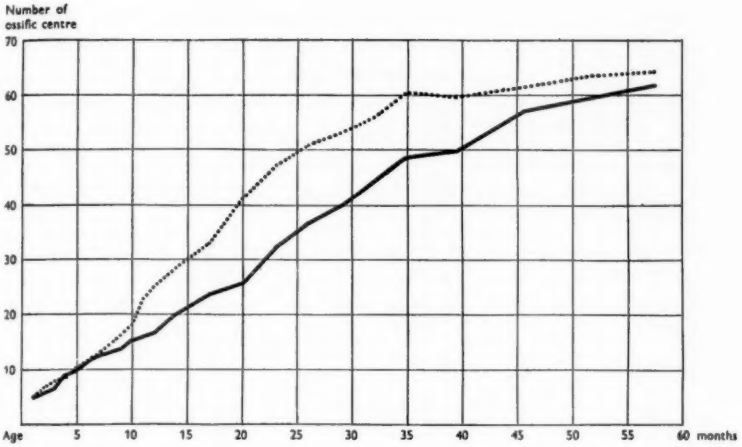


Fig. 19. Number of ossific centres at different ages. Whole line = boys, broken line = girls.

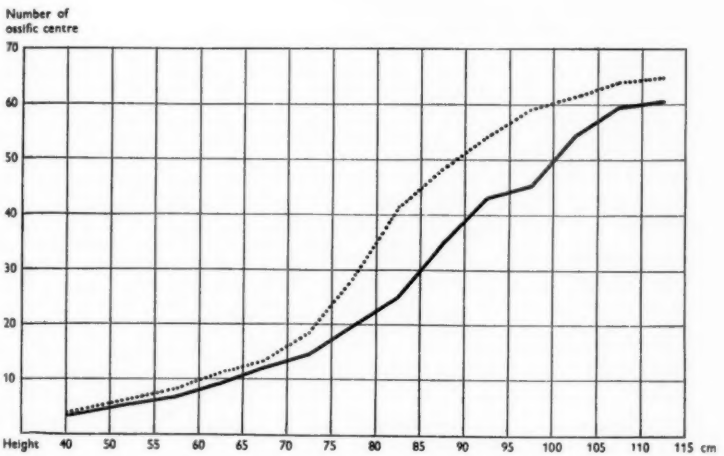


Fig. 20. Number of ossific centres at different heights. Whole line = boys, broken line = girls.

Table 23. The difference between the number of ossific centres in girls and boys in per cent of the respective means at different ages.

Age, months	Mean difference in months between girls and boys	In per cent of the mean		Age, months	Mean difference in months between girls and boys	In per cent of the mean	
		Boys	Girls			Boys	Girls
1	−0.1	1.5	1.5	13—15	+ 8.7	44.1	30.6
2	+0.5	9.9	9.0	16—18	+ 9.4	40.3	28.7
3	+1.1	16.9	14.5	19—21	+15.8	61.8	38.2
4	+0.4	4.5	4.7	22—24	+14.9	46.3	31.6
5	+0.6	6.4	6.0	25—27	+14.0	38.1	27.6
6	+0.3	2.8	2.7	28—30	+13.4	33.6	25.1
7	+0.4	3.2	3.1	31—33	+11.7	26.5	20.9
8	+1.6	12.2	10.9	34—36	+12.0	24.7	19.8
9	+2.7	20.3	16.9	37—42	+10.0	20.2	16.8
10	+2.9	19.3	16.2	43—48	+ 4.8	8.4	7.7
11	+6.9	44.1	30.6	49—54	+ 4.2	7.1	6.6
12	+8.6	51.9	34.2	55—60	+ 2.4	3.9	3.7

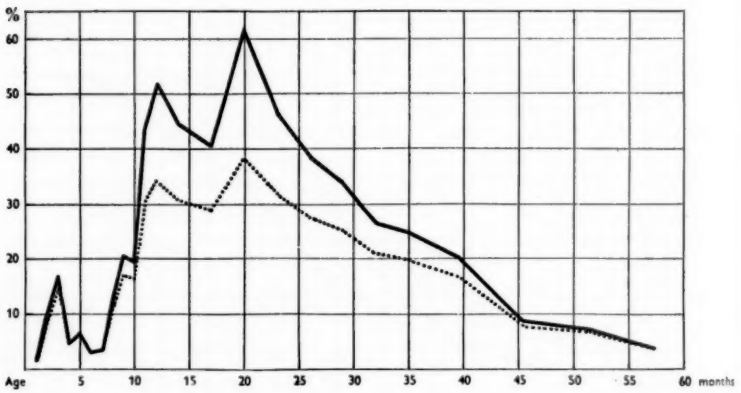


Fig. 21. The difference between the number of ossific centres in girls and boys in per cent of the respective means at different ages. When the means of the boys are used for computing the percentage, this is marked with a whole line. If the means of the girls are used, this is marked with a broken line.

Table 24. The difference between the number of ossific centres in girls and boys in per cent of the respective means at different heights.

Height, cm	Mean difference in months between girls and boys	In per cent of the mean	
		Boys	Girls
<50	+ 0.4	12.1	10.8
50— 55	+ 1.0	18.5	15.6
55— 60	+ 1.6	23.9	19.3
60— 65	+ 2.1	23.1	18.8
65— 70	+ 1.5	12.4	11.0
70— 75	+ 4.1	28.3	22.0
75— 80	+ 8.7	43.9	30.5
80— 85	+16.3	65.2	39.5
85— 90	+13.3	38.2	27.7
90— 95	+10.9	25.2	20.1
95—100	+14.2	31.5	23.9
100—105	+ 7.3	13.5	11.9
105—110	+ 4.7	7.9	7.3
110—115	+ 4.4	7.3	6.8

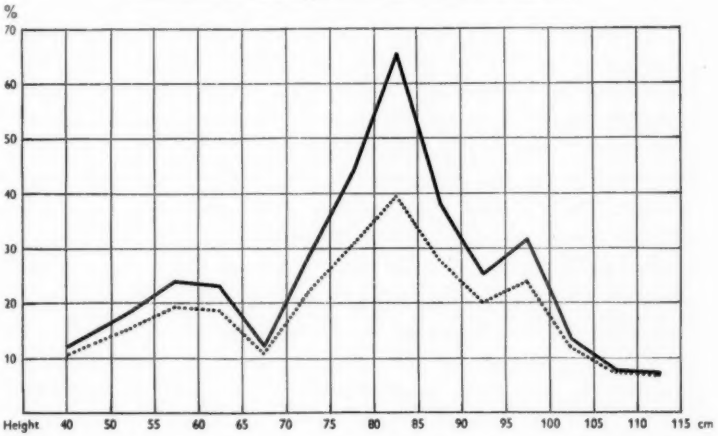


Fig. 22. The difference between the number of ossific centres in girls and boys in per cent of the respective means at different heights. When the means of the boys are used for computing the percentage, this is marked with a whole line. If the means of the girls are used, this is marked with a broken line.

Summary.

In previous investigations on the development of the ossific centres the practice has generally been to determine the differentiation of the centres from the observations made on the bone development taking place in *one* area of ossification, usually the hand or hand and foot. In the present study, the author shows that there is no absolute correlation between the appearance of the different ossific centres, and that it is consequently not possible to draw hard and fast conclusions regarding other centres on the basis of the differentiation present in a few isolated areas. Not even in the two halves of the body do the corresponding centres make their appearance quite uniformly.

In order to establish a firmer foundation for judgments on the development of the ossific centres, the author has studied by means of a number of simultaneous observations the dates of appearance of the centres in several readily accessible ossification areas; in other words, the skeleton of all the extremities on the right side of the body, comprising 68 ossific centres, and in a few subjects also the corresponding parts of the skeleton on the left side, for purposes of comparison. The age of five years was taken as the upper age limit, because there are only a few centres which appear in these areas after this age. A total of 1 190 observations were made on 852 individuals, and the material was then studied from the statistical standpoint.

The investigation brought out the fact that there is great variation in the times of appearance of the individual ossific centres, and also that there is a considerable difference between the sexes, the centres of the girls differentiating much earlier than those of the boys. Correlation calculations proved that there are significant and positive correlations of equal degree between

the differentiation of the ossific centres and age, height, and weight. By means of partial correlations between age and height, and ossific centres, it was established that in infancy the height shows a stronger correlation with ossific centre development than age does, while later on, the correlation is largely equal.

Normal tables for practical use were constructed. Since, as was mentioned before, the correlation between height and appearance of ossific centres is more marked in the first years of childhood and later on is of the same magnitude as the age correlation, only the differentiation present at different heights may, for the sake of simplicity, be used when judging the stage of development (tables 21—22, figs. 17—18). In uncertain cases, the age tables can also be used for comparison (tables 19—20, figs. 15—16).

References.

ADAIR, F. L. and SCAMMON, R. E.: Am. J. Obst. and. Gynaec. 2: 35, 1921. — ALEXANDER: Arch. f. Physic. Medix. u. med. Techn. 1: 108, 1906. Cited by Fujinami. — BADE, P.: Fortschr. a. d. Geb. d. Röntgenstr. III: 138, 1899—1900. — BAYER, L. and NERWELL, R.: Endocrinology, p. 799, 1940. — BEHRENDSEN, E.: Deutsche med. Wschnschr. 27: 433, 1897. — BIRCHER, E.: Fortschr. a. d. Geb. d. Röntgenstr. 21: 302, 1909. — ——: Archiv f. klin. Chirurgie, 91: 554, 1910. — BOGAN, J.: Am. J. Dis. Child. 59: 806, 1940. — BORNHARDT, A.: S:t Petersburger mediz. Wschnschr. 48: 1888. Cited by Åkerlund. — BROCK, J.: Biologische Daten f. den Kinderarzt, 1931, Berlin. — BROMAN, I.: Människans utveckling före födelsen, p. 277, 1927. — BROMAN, B., DAHLBERG, G., LICHTENSTEIN, A.: Acta Pæd. XXX: 1, 1942. — BUEHL, C. C. and PYLE, S. I.: J. Ped. 21: 335, 1942. — BULLITH, C.: Am. J. Roentgenol., 20: 548, 1928. — BURKE, E. and STUART, H. C.: J. Ped. 12: 493, 1938. — BUXTON, E. and KNOX, R.: Brit. J. Radiol. 32: 199, 1927. — CAMP, J. D. and CILLEY, E. I. L.: Am. J. Roentgenol., 26: 905, 1931. — CAFFEY, J.: Pediatr. X-Ray Diagnosis, 1945, Chicago. — CHRISTIE, A. U., DUNHAM, E. C. D., JENSS, R. M. and DIPPEL, A. L.: Am. J. Dis. Child. 61: 471, 1941. — CLIFT, L.: Am. J. Radiol., 12: 23, 1924. — DAHLBERG, G.: Zeitschr. f. Morphologie u. Anthropologie, 29: 288, 1931. — ——: Statistical Methods for Medical and Biological Students, 1940, London. — DANIELS, A. L.: Am. J. Dis. Child. 62: 279, 1941. — DOMMISSE and LEIPOLDT, Cited in Am. J. Dis. Child. 54: 590, 1937. — DUNHAM, E. C., JENSS, R. M., CHRISTIE, A. U.: J. Ped. 14: 156, 1939. — ENGEL, S. u. SCHALL, L.: Handbuch der Röntgen-Diagnostik u. Therapie im Kindesalter, 1933, Leipzig. — FLECKER, H.: Am. J. Roentgenol. 47: 97, 1942. — FLORY, J.: Soc. Rec. Child. Developm. 1: 141, 1936. — FRANCIS, C. C.: Am. J. Dis. Child. 57: 817, 1939. — FRANCIS, WERLE, and BEHM: Am. J. Phys. Anthropol. 24: 273, 1939. — FRIEDENTHAL, H.: Erg. inn. Med. 9: 505, 1912. — ——: Ibid. 11: 685, 1913. — FUJINAMI, K.: Fortschr. a. d. Geb. d. Röntgenstr. XVII: 249, 1911. — GABINUS, O.: Acta Pæd. 25: 59, 1939. — GRALKA, S.: Röntgendiagnos im Kindesalter, 1927, Leipzig. — GRASER, E.: Zeitschr. f. Kinderheilk. 60: 30, 1938. — GÖRTZ, G. and GÖRTZ, S.: Bibliotek f. læger, 159: 43, 1937. — HAHN, H.: Festschr. z. 70. Geburtstag von Carl v. Kupffer, p. 731, 1899. — HASSELWANDER, A.: Zeitschr. f. Morphologie u. Anthropologie V: 438, 1903. — ——: Anatom. Anzeiger, XXXII: 608, 1908. — ——: Zeitschr. f. Morphologie u.

Antropologie, XII: 1, 1909. — HELMANN u. POTPENSCHNIGG: Jahrb. f. Kinderheilk. p. 65, 1907. — HODGES, J.: Am. J. Roentgenol. 30: 809, 1933. — HOLMGREN, I.: Nord. Med. Ark. 11. no. 5: 1, 1909. — ——: Nord. Med. Ark. 11, no. 1: 181, 1910. — INGALLS, E.: Endocrinology, 20: 710, 1941. — IZIKOVITCH, G.: Fortschr. a. d. Geb. d. Röntgenstr. 54: 249, 1936. — JOSEFSON, A.: Svenska Läkarsällskapets handlingar, 1915. — KÖHLER, A.: Grenzen des Normalen und Anfänge des Pathologischen im Röntgenbilde. 1928, Leipzig. — LINDERS, F. J.: Om sambandet mellan nyfödda barns längd och vikt och moderns havandeskapstid, 1930, Stockholm. — MACARTHUR, P.: Arch. Dis. Child. 11: 243, 1936. — MAC NAHIR V.: Am. J. Dis. Child. 58: 295, 1939. — MARESH, B.: Am. J. Dis. Child. 66: 227. 1943. — MENESS, T. O. and HOLLY, L. E.: Am. J. Roentgenol. 28: 389, 1933. — MOLITCH and POLIAKOFF: Endocrinology, 20: 816, 1936. — MONRAD, S.: Ugeskr. f. Læger, 96: 215, 1934. — NOBÉCOURT, P. et KAPLAN, M.: Traité de Médecine des Enfants, 1934, Paris. — NYLIN, G.: Acta Med. Scandinav. suppl. XXXI, 1929. — v. PFAUNDLER: Zeitschr. f. Kinderheilk. 14: 1, 1916. — v. PIRQUET: Ibid. 6: 253, 1913. — ——: Ibid. 36: 63, 1923. — PRYOR, J.: J. of anat. 62: 499, 1928. — v. RANKE, H.: Münch. m. Wochenschr. 43: 1365, 1898. — RAUBER-KOPSCH: Anatomie des Menschen, 1938, Leipzig. — RIEDER-ROSENTHAL: Lehrbuch der Röntgenkunde, Bd. 2: 187, 1918. — ROBINOW, N.: Am. J. Dis. Child., 64: 229, 1942. — RUCKENSTEINER, E.: Radiologische Praktika, Bd. XV, 1931, Leipzig. — SAWTEL, E.: Am. J. Dis. Child., 37: 61, 1929. — SCHINZ, G.: Lehrbuch der Röntgendiagnostik, 1928, Leipzig. — SCHIÖTZ: Sv. Läkartidningen, 27: 776, 1930. — SCHMID-MONRAD: Jahrbuch f. Kinderheilk. 40: 84, 1896. — SHANDS, P.: J. of bone and joint surg., 18: 824, 1926. — SHELTON, K.: J. A. M. A. 96: 759, 1931. — ——: Endocrinology, 17: 667, 1933. — SIEGERT, F.: Atlas der normalen Ossification der menschen Hand. — SONTAG, L. W.: Am. J. Dis. Child. 56: 300, 1938. — — —: Ibid. 56: 1248, 1938. — SONTAG, SNELL and ANDERSON: Am. J. Dis. Child. 58: 949, 1939. — STETTNER: Arch. Kinderheilk. 68: 342, 69: 27, 1921. — ——: Zeitschr. f. Kinderheilk. 51: 435, 52: 1, 1931. — STUART, H. C.: J. Ped. 5: 194, 1934. — STUART, H. C. and VICKERS, V.: Am. J. Dis. Child. 52: 753, 1936. — v. SYDOW, G.: Acta Pædiatr. XXVIII: 8, 1941. — TODD, T. W.: Am. J. Dis. Child. 55: 149, 1938. — WILBAR, CH. L.: J. Ped. 21: 504, 1942. — WILKINS, L.: J. Ped. 12: 429, 1930. — WILKINS, BLOCK and LANDSBERG: Am. J. Dis. Child. 56: 461, 1938. — WILMS und SICK: Fortschr. a. d. Geb. d. Röntgenstr. Erg. Bd. 9, 1902. — VOGT, E. C. and VICKERS, V.: Radiology, 31: 441, 1938. — v. WYSS, R.: Fortschr. a. d. Geb. d. Röntgenstr. 111, 1899—1900. — YDKIN, A.: J. Ped. 12: 701, 1938. — YLPPÖ, A.: Ref. Zeitb. Kinderheilk. 12: 380. — ZUCK, T. T.: J. Ped. 3: 424, 1933. — ÅKERLUND, Å.: Fortschr. a. d. Geb. d. Röntgenstr. Erg. Bd 23, 1918.

FROM THE CHILDREN'S HOSPITAL MEDICAL DEPARTMENT, GÖTEBORG;
THE CENTRAL LABORATORY OF SAHLGREN'S HOSPITAL, GÖTEBORG;
AND THE PEDIATRIC CLINIC AT NORRTULL HOSPITAL, STOCKHOLM

A Study of the Development of Rickets in Premature Infants

by

GERT v. SYDOW

ACTA PÆDIATRICA, VOL. XXXIII: SUPPLEMENTUM II

Victor Pettersons Bokindustriaktiebolag
Stockholm 1946

Table of contents

	Page
Preface	7
Introduction	9
The Material	18
Methods	22
A. Chemical methods	22
B. Röntgenological examination	24
C. Statistical methods	25
Chemical determinations	26
I. Normal values	26
II. Premature infants fed human milk without any supplement	40
III. Premature infants fed human milk + vitamin D	50
IV. Premature infants given cow's milk but no vitamin D	57
V. Premature infants given cow's milk + vitamin D	62
VI. Premature infants given different supplies of mineral preparations	67
VII. A comparison of the serum values in the different groups	72
Röntgenological examinations	77
I. Occurrence of different röntgen signs	77
II. Serum values at röntgen signs of rickets	101
Discussion	111
Summary	116
References	119

Preface

About six years ago, Professor *Arvid Wallgren,* who was then my chief, suggested that I should try to investigate the difference in vitamin D demands between breast-fed and bottle-fed infants. I decided to study this question in premature infants and began by comparing pairs of twins of which the one was fed human milk and the other cow's milk, but no vitamin D. Surprisingly enough, the twin who had had human milk in almost all cases showed more abnormal values for serum phosphatase and serum inorganic phosphorus, than the one who had had cow's milk, and also in some cases röntgen signs of rickets, while the cow's milk twin showed normal röntgen findings. These observations gave rise to this investigation.

Professor Wallgren has been most interested in my work all the time, and facilitated it in every way. His help and encouragement have been of the greatest importance to me, and I am most grateful to him for this.

The chemical analyses have all been carried out at the Central Laboratory of Sahlgren's Hospital. To Professor *Jörgen Lehmann,* chief of the Laboratory, I am much indebted for the hospitality and interest he has shown me during these years and for his readiness to discuss the different problems which have suggested themselves during the work and which have on many points decided the course of the investigation.

The chief röntgenologist of Sahlgren's Hospital, Med Doctor *Gösta Runström,* has earned my warm thanks for the numerous röntgen pictures and for his willingness to arrange the röntgen examinations in the best way for the premature infants. The chief röntgenologist of the Children's Hospital in Göteborg, Dr. *Carl Johan Hansson,* and the röntgenologist of the Norrtull Hospital in Stockholm, Dr. *Oliver Axén,* have both examined all the röntgen pictures. I am most grateful to them for their interested work in this matter.

To the present Chief of the Children's Hospital Medical Department, Med. Doctor *Yngve Åkerrén,* and to the Chief of the Obstetric Department of Sahlgren's Hospital, Professor *Emil Jerlov,* I wish to express my gratitude for the good conditions under which I have worked and for their kindly interest.

To my colleagues at the Children's Hospital and Norrtull Hospital I express my thanks for good comradeship and much help in my work. Especially would I thank my friend Dr. *Herbert Nathhorst* for an unforgettable symbiosis during the last months of scientific labour.

In carrying through such an investigation as this it is of very great importance that the nurses who tend the children and distribute the different foods and preparations are themselves interested in the work and willingly follow the directions given in connection with it. I would especially thank the chief nurse of the premature ward Miss *Anna-Greta Mård-Banck* who has made great and self-sacrificing efforts for my work and reported many valuable observations. Her share in this production has been very great.

Mrs. *Helga Larsen* has carried out most of the chemical analyses and also made a great part of the statistical calculations. Her skilful and conscientious work, good judgement and intelligent criticism have been indispensable for the investigation. I thank her warmly for all her efforts and her interest in the matter. Also to the nurses at the Central Laboratory I am indebted for much and willing help.

The translation has been done by Miss *Agnes Dawson* and I thank her for her unsparing efforts and good work.

For the investigation I have received financial help by generous grants from the drug factory *Astra,* Södertälje, from *Göteborgs Läkaresällskap,* and from the Foundation *"Therese och Johan Anderssons minne"* and to them all I am greatly indebted.

Göteborg, 20/4 1946.

GERT V. SYDOW

Introduction

The present conception of the development of rickets has been summarized by Barnes (1945) in a way fairly universally accepted: "Rickets very definitely is a systemic disease which has as its direct causative agents either a lack of adequate vitamin D or insufficient calcium and phosphorus. Supply adequate amounts of minerals and vitamin D and the disease disappears." Most authors, however, make a definite division between human rickets on the one hand, and rickets in some species of animals on the other hand. In human rickets, the main cause is a lack of vitamin D, while for example in experimental rickets in rats, the main cause is an insufficient supply of calcium and phosphorus, or, more often, a faulty calcium-phosphorus ratio in the food. In the latter case, the element most scantily supplied will to a great extent be precipitated in the intestine as insoluble calcium phosphate and there will then be an insufficient absorption of this element.

To produce experimental rickets in rats, the animals have usually been given a food with an excessively abnormal calcium-phosphorus ratio. Shohl (1936) has shown, however, that this can also be done with a normal ratio (between 2: 1 and 1: 2) if the quantitative supply of calcium and phosphorus is sufficiently small. In some other species of animals (e.g. different ruminants and pigs: Du Toit and Malan 1937) it has also been possible to produce rickets by means of an excessively abnormal calcium-phosphorus ratio, and this in spite of a rich supply of vitamin D. In others, e.g. monkeys (Gerstenberger 1938) rickets has been produced even with a normal ratio and has been cured after a supply of vitamin D.

In infants the possibility has generally been rejected that the calcium-phosphorus ratio can play any part in the development of rickets, since they usually obtain all their supply of calcium and phosphorus in the form of milk, in which the ratio must be regarded as lying within normal limits whether it is cow's milk or human milk. Nor has any role been generally attributed to an insufficient supply of calcium and phosphorus, partly because breast-fed infants undoubtedly have rickets less often, or at least less severely than bottle-fed infants, in spite of the fact that the calcium and phosphorus content of the ordinary cow's milk formulas is much greater than of human milk. Instead of this it is usually thought that, most often in consequence of a lack of vitamin D, the absorption or retention, or both, of the bone-building minerals has deteriorated so much that in spite of a sufficient supply there arises a lack of these materials in the ossification centres. Holt and McIntosh (1939) called such rickets "metabolic rickets" to differentiate it from the "growth rickets" which arises in experimental animals because the external supply of building materials is too small for the growing skeleton.

However, voices have not been lacking that call attention to the small mineral supply in human milk in relation to the great rapidity of growth, especially in premature infants. Thus Ylppö (1919) stated that "die Frauenmilch — die ja für ein ausgetragenes Neugeborenes für die erste Lebenszeit die optimale Nahrung darstellt, auch für die Frühgeburten die einzige ist, mit welcher wir die besten Erfolge erzielen können. Aber hiermit ist keineswegs gesagt, dass sie eine ideale Nahrung für die Frühgeburten wäre. Wir müssen uns vielmehr vorstellen, dass der geringe Salz- und Eiweissreichtum der Frauenmilch nicht genügt um den Bedarf des ausserordentlich intensiven Wachstumtriebes des Körpers, vor allem den der Knochen, in dieser Zeit zu decken. So muss man z. B. annehmen dass die geringen Mengen von Kalk, die dem Kinde in der Frauenmilch zugeführt werden, nicht genügen um die Ansprüche des wachsenden Skeletts zu befriedigen. — Die Salzarmut in der Frauenmilch hemmt vermutlich bei den Frühgeburten aber nicht nur das

normale Wachstum, sie führt allem Anscheine nach mit der Zeit auch zu wirklichen krankhaften Prozessen in den Knochen, die ihrerseits dann in besonderer Weise auf den normalen Wachstumstrieb hemmend wirken. Ich meine damit die Rachitis."

Numerous analyses of the foetus at different stages of development have shown that the mineral content of the body rises continuously during the whole of the foetal life, but that the rise is especially great during the last months of gestation. According to the investigations of Givens and Macy (1933) and Iob and Swanson (1934), Smith (1945) calculates that the foetus between 4 and 6 lunar months increases its calcium content from 1.0 to 3.7 gms, between 6 and 8 lunar months by 5.2 gms to 8.9 gms, and between 8 and 10 lunar months by 12.1 gms to 21.0 gms. "Although these are averages from data which show a surprising scatter at the time of birth it is obvious that more than half the acquisition occurs during the last fifth of gestation." "In general, the increase of phosphorus in the fetal body follows a curve like that for calcium, rising (though less steeply) during the last months of gestation." (Smith 1945.) It seems therefore probable that most premature infants are born with a considerably less mineralized skeleton than full term infants.

After birth the infant is supplied with mineral substances for its growth exclusively through the milk it consumes. The mineral content of human milk seems to vary considerably, but nearly all investigators have found the average calcium content to be in the neighbourhood of 30 mg, and phosphorus content in the neighbourhood of 15 mg per 100 ml. For cow's milk the average has generally been found to be about 120—130 mg for calcium and 90—100 mg for phosphorus per 100 ml. Of this only a part is retained. In order to maintain during growth the mineral content per kg body weight at birth in the full term infant, Leitch (1937) considers that a calcium retention of 0.130 gm daily during the first month of life, 0.310 during the second and 0.250 gm during the third month are necessary. Actually, however, a breast-fed infant only retains 0.082, 0.085, 0.102 gm calcium daily during the first three months of life respectively, and the skeleton thus gradually becomes more and more lacking in cal-

cium. An infant fed undiluted cow's milk, on the other hand, retains during the second month 0.417 and during the third month 0.465 gm calcium daily. In comparing prolonged metabolic assays of a new-born infant fed human milk and one fed cow's milk, Swanson (1932) found that the former for the first three months retained daily 0.10 millimols calcium and 0.81 millimols phosphorus, while the latter of the same age and with a similar growth rate, retained 2.87 millimols calcium and 2.97 millimols phosphorus. During the second three months' period, after a dose of cod-liver oil had been added, the breast-fed infant retained daily 1.79 millimols calcium and 1.48 millimols phosphorus while the cow's milk infant retained 7.28 and 4.47 millimols respectively.

The mineral content of premature infants fed human milk alone decreases still more drastically (e. g. Hamilton 1922). "It is not surprising that prematurely born infants who have been fed only human milk almost invariably develop rickets." (Stearns 1939). The most likely cause of this is the fact that the premature infants, who at the start have a lower mineral content than the full term, after birth also consume smaller amounts of human milk in proportion to their growth rate than full term infants during the first period of life. The reason cannot be that they have less capacity to retain the minerals supplied. Benjamin et al. (1943) found that premature infants when given cow's milk and a large dose of vitamin D had on the contrary greater capacity for retaining calcium and phosphorus than full term infants with the same feeding. This they explained was because the premature infants who had a rich supply of minerals, at the same time as they satisfied their need of minerals for growth, also sought to supplement their originally insufficient stores of these materials, which the full term infants did not need to do. In contrast to this they found that premature infants fed human milk plus vitamin D showed a much lower retention of calcium than those fed cow's milk plus vitamin D, not only in total quantities but also in proportion to the supply. The breast-fed infants thus retained only 45 % of the calcium provided by the human milk, while the cow's milk infants retained 71 % of their consi-

derably greater supply. On the other hand, the breast-fed infants retained almost all (87 %) the phosphorus supplied, as against 61 % for the cow's milk infants. As an explanation of this remarkable fact, the authors stated that the breast-fed infants must use a large part of their small phosphorus supply for building up protoplasm instead of skeleton, and that therefore no more calcium could be retained than corresponded to the amount of phosphorus available for bone-building.

Since the decreasing mineral content in the body during breast-feeding has been demonstrated even in full term infants, it is not astonishing that this fall is considered by many to be normal, while the high mineral content which continues later in cow's milk-fed infants is termed "Supermineralisation" (Rominger and H. Meyer 1927), although the mineral content even in these latter does not exceed very considerably the level at birth (Leitch 1937). Beumer (1942) considers that "da die natürliche Ernährung bei niedrigstem Mineralangebot und geringstem D-Bedarf den besten Schutz gegen Rachitis gewährt, ist erwiesen, dass die sich dabei stärker ausbildende, physiologische Osteoporose unter normalen Verhältnissen zur Rachitis in keiner Beziehung steht." As regards the high mineral content in infants fed cow's milk, he admits that "ungünstige Folgen dieser Supermineralisation wurden noch nicht beobachtet, doch ist denkbar, dass in Zeiten der Not ein höherer Mineralvorrat von Vorteil sein kann. Gelangen Brustkinder, vor allem solche, die übermässig lange gestillt wurden, oder mit starken Milchverdünnungen ernährte Säuglinge unter ungünstige Umweltsbedingungen, so entwickelt sich gerade bei ihnen infolge der geringen Mineralreserven nicht selten ein Rachitistyp von ausgesuchter Schwere mit hochgradiger Osteoporose, Frakturen und Infraktionen und tief gesenkten Ca-Serumwerten." Most authors do not, however, make this admission. "Retention of calcium and phosphorus is lowest with breast milk feeding and highest with an intake of cow's milk plus vitamin D. Although retention may be multiplied many times with such a dietary shift, there is no evidence that a superior type of bone results from this 'super-mineralization'" (Smith 1945). — Eliot and

Park (1945) who consider that "all diets contain ample phosphorus, but not ample calcium, according to European and American standards, provided milk, the only food rich in calcium, is lacking," make no exception for human milk but instead make the reservation that "a hypothetic defect in the diet is the presence of calcium and phosphorus in such combination as to make utilization by the body difficult when the antirachitic ultra-violet radiations are deficient. It seems possible that in cow's milk such may be the case." — Barnes (1945) has almost the same opinion, but he admits that "too little milk may make an insufficient amount of calcium and phosphorus available, as in the premature baby with its relative small food intake, and predispose towards the development of the disease."

A series of factors other than a low mineral supply has been put forward to explain the fact accepted by all that premature infants have a considerably greater disposition to rickets, and that they get it earlier and more severely than others. Crosse (1945) lists the following causes:

1. "They have a deficient store of calcium, phosphorus and vitamin D at birth.

2. A relatively small part of the skeleton is calcified at birth.

3. Their absorption of fat, vitamins and mineral salts is defective.

4. Their basal metabolism is low.

5. Their growth is relatively more rapid.

6. Because of their small size and general frailty they are less exposed to natural sunlight.

7. The increased incidence of infection and digestive disturbances reduces still further the poor absorption of vitamins and mineral salts."

To this can be added Mai's hypothesis (1936) of a "körpereigene mitogenetische Ultraviolettstrahlung" as he considered he had proved in healthy normal infants but could not prove in premature infants, nor in those with active rickets. Ylppö

(1931) assumed that "die Hauptursache zur Entstehen der Rachitis muss aber in der Unvollkommenheit des gesamten Organismus der Frühgeburten erblickt werden." He specially emphasized the poor capacity of the premature infant to digest milk-fat, even from human milk, as it is excreted through the bowel in the form of lime soaps and in this way calcium is lost. Finally Marfan (1941) has set up a hypothesis of his own regarding the pathogenesis of rickets, according to which it is only partly an avitaminosis D, it demands an infection or an intoxication to bring it to action. "L'appauvrissement de l'organisme en ce principe (la vitamine D) permet aux infections et intoxications de déterminer les réactions osseuses qui aboutissent au rachitisme."

When it is a question of translating the knowledge of the development of rickets into prophylactic and therapeutic treatment, nearly all the new text-books recommend only a supply of vitamin D. To make sure that the minerals supplied are utilized to a maximum, premature infants are prescribed larger doses of vitamin D than are generally given to normal infants. In this respect Malmberg (1942) e. g. found that for premature infants with a birth weight up to 2 000 gms the need of vitamin D in continuous treatment with short interruptions was about 10 000 I. U., possibly a little lower, but certainly above 6 000 I. U. His patients however have not all been reared exclusively on human milk. T. Jonsson (1945) and Klackenberg (1945) in investigations of premature infants found that a big dose of vitamin D of 500 000 or 600 000 I. U. given in the neonatal period gives relatively good protection against rickets.

After Hamilton and Schwartz (1933) had shown that experimental rickets could be prevented by a supply of organic acids which produced an intestinal acidity but a metabolic alkalinity, attempts were made in some quarters to apply this method to infants, and success has been achieved in improving the utilization of the minerals in cow's milk and preventing or curing rickets in this way. (Shohl and Butler 1939, Malmberg 1942, Rominger 1944.) Eliot and Park (1945) recommend this method in certain special forms of rickets.

In the older text-books, however, as a remnant of views prevalent before the vitamin era, instructions can be found to use extra supplies of minerals as a prophylaxis for rickets. Ylppö (1931) recommended that premature infants should have, in addition to human milk and other careful treatment with the avoidance of infections, a supply of extra calcium e. g. in the form of calcium phosphate. He emphasizes however, "dass es eine noch offene Frage ist inwieweit man durch die künstliche Zufuhr von Kalk die Resorption bei den Frühgebuhrten wesentlich zu erhöhen vermag." — Gerstenberger and Nourse (1926) found that rickets could be prevented in premature infants with a very small dose of cod-liver oil, provided that it is administered as soon as food is consumed, and that the food contains a higher mineral and protein content than does human milk. If human milk is used it should be supplemented with boiled skim milk. — Gerstenberger (1939) was finally able to make rickets in infants heal and disappear after parenteral injections of phosphate solutions.

In the most recent years some authors have again come to the conclusion that human milk is unsatisfactory as the sole food for premature infants. Benjamin et al. (1943) state as a consequence of their above-mentioned experiments "that from the stand-point of skeletal formation, breast milk, even in the presence of sufficient vitamin D, was not the food of choice for a premature baby unless it was supplemented by extra calcium and phosphorus." — Tow (1942) states that in his experience simple milk mixtures have given much better results than breast milk in rearing premature infants. But he is probably thinking not so much of a prophylaxis against rickets as of maintaining a maximum weight gain. Finally Crosse (1945) advises that "in addition to the administration of vitamin D extra calcium and phosphorus should be given. This is particularly necessary if the infant is breast-fed, as it is suggested that the small amount of phosphorus in breast milk limits the retention of calcium." She recommends Syrup of calcium lactophosphate, in a dose probably corresponding to about 1.3 mg calcium and 1.0 mg phosphorus per kg body weight.

No investigations seem to have been published on whether premature infants fed only human milk get rickets more or less easily than premature infants reared in another way, or whether by an extra supply of mineral the rickets risk is lessened for premature infants fed human milk.

The object of this work is to investigate whether there is a type of rickets in man, primarily in premature infants, which depends only or mainly on a scarcity of minerals, and if so, whether it can be prevented or cured by supplying minerals or in another way.

The Material

The material consists of those premature infants who during the years 1943 and 1944 and the first half of 1945 were born at the Obstetric Department of Sahlgren's Hospital and were tended there in the premature ward, whose birth weight did not exceed 2 000 gms and who were considered able to be subjected to the necessary examinations without risk. To complete a few series a small number of infants were examined also during Autumn 1945.

The reason for taking only infants with a birth weight up to 2 000 gms is that only a limited number of blood analyses could be made at a time. More frequent opportunities could thus be expected of observing the appearance of rachitic symptoms in the smallest infants, partly because in them rickets would probably appear earlier on the average, partly because the observation time in the hospital would probably be longer than in the bigger premature infants.

No selection was made, except that, as already stated, the most debilitated were excluded in consideration of the risk of cyanosis attacks in connection with the examinations. However, the first examinations of the most debilitated infants included undoubtedly took place somewhat later than of the more vigorous, so that the latter in general were examined during the first week of life, the former often during the second week or still later. This can have had some influence on the difference between the results obtained from the earliest age periods.

Before the first examination all the infants were fed exclusively human milk in quantities according to the rules practised in the ward for their size. The human milk had come partly from

the infant's own mother and in that case was given raw, and partly from the mothers' milk centre and then was boiled. Practically all the infants, at least during the first period of life, had human milk from both these sources. Besides the human milk the infants were given water, sometimes also Ringers solution parenterally. All the infants were, of course, given vitamin K at birth, but otherwise no extra vitamin supply was given before the first examination. As all the beds in the premature ward are well covered with blankets during the short time the windows are open, no effect of ultra-violet rays need be taken into account either before or during the assay period.

Directly after the first examination the infants were put on the diet to be tested. The diet of the infant was determined before its birth, in that for a certain period every second or every third infant had the same food. In this way an attempt was made to avoid any subjective choice and to make the assay series comparable. In many cases, however, after some time the feedings of two comparable infants were exchanged. This was especially done with twins.

During the assay period the infants were examined with blood analyses and röntgen at regular intervals, in most cases every fourteen days, sometimes every week, and sometimes once a month. They were also subjected every week to a careful clinical examination. The supply of food, weight conditions, unhealthy changes etc. were recorded daily.

The infants were kept in the premature ward during the assay period. Among the oldest however, a small number of examinations were made on infants who had some time previously been discharged to their homes or to an Infants' Home where they lived under well-known conditions, and who returned for control examination.

For the statistical analysis the infants are grouped according to their birth weight, and the chemical and röntgenological examination results are grouped according to their age at the examination.

Table 1 shows this distribution of the material.

The t w i n s included in the material for this analysis have

Birth weight	Number of infants examined	No. of examinations of the different age groups (days)									Total no. of examinations
		1–3	4–6	7–15	16–30	31–45	46–60	61–75	75–105	106-180	
1010—1500	11	—	2	5	7	6	7	7	13	8	55
1510—1750	45	11	17	16	38	32	27	22	26	18	207
1760—2000	62	26	17	19	52	59	33	26	21	29	282
1010—2000	118	37	36	40	97	97	67	55	60	55	544

Table 1. Distribution of the material according to birth weight and age when examined.

been included with the other premature infants of the same weight class. They will however also be included in a report on a large series of premature twins collected at the same time as this series. In this second report pairs of twins with different food will be compared. In this way one can, better than in the material at present under discussion, exclude any differences between the groups due to hereditary factors, the mothers' prenatal care etc. and refer with greater certainly the differences found to the different food. The material of the twins will be reported in a later work.

Normal material

To get an idea of the normal values of the chemical methods used in the investigation two normal groups have been examined.

One normal group consists of *new-born infants* with a *birth weight between 3 000 and 4 000 gms* examined during the first week of life. From the 7/11 1944 to the 10/7 1945 the nurse in one of the obstetric wards chose every Tuesday a baby born on each of the six previous days. The infants had to have been fed exclusively human milk and neither they nor their mothers should have shown any important sign of ill health. The children were not born of mothers in poor social conditions.

This group came to consist of 130 children. Of the mothers two were under 20 years old and one over 40, 42 were between

31 and 40 years old. 57 were uniparous, 43 bi-parous; the other 30 had previously gone through more than one pregnancy. 107 of the mothers had had prenatal care for a least three months, but only 77 had taken any preparation containing vitamin D for more than a month.

The other normal group consists of children between *2 weeks and six months old* with a *birth weight over 3 000 gms,* who had been examined at a childrens' welfare centre during the time 15/6 1944 to the 14/6 1945. They had to have been fed exclusively human milk (a few cases, however, had been given for a short time at least a month before the examination a small addition of cow's milk) and during the period October to May to have been given an additional daily dose of 1 000—1 500 I. U. of vitamin D. Their home conditions had to be considered satisfactory. They had to have been well since birth, and at the examination should show good general condition and no important symptoms of illness.

This group consists of 144 infants of whom 121 were between 16 and 105 days old. Only three of their mothers had had no prenatal care at all. 60 of the infants were the first children, 49 the second children, 21 had more than one sister or brother, and for 14 there was no information as to the number of their siblings.

As a by-product a small group was obtained which consisted of 18 infants aged 2 weeks—6 months (12 of them aged 46—75 days) who fulfilled the same demands as the above except that they had been *fed well-composed cow's milk formulas* instead of or as a supplement to human milk.

Methods

A. Chemical Methods

The blood for the analyses was obtained by heel puncture. Usually 2 1/2 cc. blood was enough for double determinations of all the analyses, but from the new-born with hemoglobin values higher than about 130 a little more had to be taken because of the scanty serum formation in such blood. To get an ample blood flow the heel was warmed in water before the puncture and afterwards cautiously milked towards the puncture. In order to prevent hemolysis in the blood care was taken that the skin was absolutely dry when punctured and, a very important thing, that the blood fell freely into the test tube which was not allowed to press against the skin, as then blood cells would be crushed and hemolysis would arise.

All the analyses were carried out on serum. Before centrifugation the blood sample had to stand for at least an hour, but not more than two hours in order to prevent the development of autolysis of organic phosphorus compounds, which would increase the serum content of inorganic phosphorus (Martland and Robison 1924, Jorpes and Magnusson 1927). At first this source of error was not considered and, as a consequence, now and then very high values for serum inorganic phosphorus were obtained. Such values have not occurred since the error was corrected, but because of it all values for serum inorganic phosphorus obtained before the 1st of October 1943 have been excluded from the statistical analysis of the results.

1. *The serum phosphatase determinations* were carried out according to the modification by I. Buch and H. Buch (1939) of

the King and Armstrong method. In order to render possible analyses on 0.1 cc. of serum instead of on 0.25 cc. as done by Buch & Buch, J. Lehmann has further modified and on certain points simplified this method. The proportions between the different solutions to be mixed are retained but the sodium hydroxide solution and the carbonate solution are combined, thus reducing the number of pipettings. The analysis is carried out as follows: — 2 cc. of the substrate-buffer are pipetted off into an ordinary centrifuge tube and heated to 37° C in a water-bath for about 5 minutes: then 0.1 cc. of serum is pipetted into the solution. After exactly 15 minutes in the thermostate, 0.9 cc. of 25 % trichloracetic acid is added. Then the tube is shaken and its contents filtered. To 1.6 cc. of the filtrate are then added 2.08 cc. of the sodium hydroxide-carbonate buffer (128 cc. 0.62 n NaOH + 80 cc. 20 % Na_2CO_3) and 0.32 cc. of the phenol reagent. The fluids are well mixed and allowed to stand for 15 minutes at 37° C until the final blue colour develops. — A control test is prepared in exactly the same way except that the trichloracetic acid is added immediately after the serum. — The reading is carried out in a Pulfrich Stuphenphotometer in 1 cm. cells using filter S 57. The test is read against a blank test. Usually all tests are read against the same blank test as these seldom differ very much, thus saving time in series analysis. The extinction value read is inserted in a diagram from which the phosphatase units are obtained, or these are calculated according to the formula

$$\text{Ph.U.} = \text{Ext.} \times 37$$

where Ph.U. stands for phosphatase units and Ext. for extinction. As the filters of different photometers can differ slightly from each other, they should be standardized with known phenol solutions prepared according to Buch and Buch. If the extinction is higher than 0.8 the reading should be carried out in 1/2 cm. cells and the value multiplied by 2.

Double determinations rarely differ from each other by more than 1 phosphatase unit. The number of phosphatase units according to this method expresses the number of mg. phenol that

will be liberated by 1.00 cc. serum in standard condition from the disodium monophenylphosphate substrate used.

2. *The Serum inorganic phosphorus* is determined on 0.05 cc. serum according to the Lundsteen and Vermehren method (1936).

3. The *Serum calcium* is determined according to the Sobel and Kaye method (1940) modified by J. Lehmann, where the analysis is carried out on 0.1 cc. serum instead of 0.2 cc. A blank value is introduced using 0.1 cc. of a 0.9 % sodium chloride solution instead of serum. When such a blank value is not used, too high values are obtained. 0.1 cc. of a 1.0 % sodium iodide solution is used instead of a 0.05 % solution as proposed by Sobel & Kaye. — The exactness of this method is found by J. Lehmann, by analysis of serum containing 10 mg % calcium. to be:

$$\sigma = \pm 0.5 \text{ mg \%}; \; \varepsilon \text{ (M)} = \pm 0.13 \text{ mg \%}.$$

4. *The serum total protein* is determined according to the Barbour and Hamilton method modified by J. Lehmann (1944). The average exactness of this method according to Lehmann is ± 0.2—0.3 % of protein when determining at 19—21° C.

5. *Calcium and phosphorus in milk* is determined after the milk has been evaporated in a water-bath and then ashed at 550—600° C, after which the ash is dissolved in hydrochloric acid, hydrolyzed for 1 hour and neutralized. From this solution the calcium is precipitated as oxalate and titrated with permanganate. The phosphorus is determined colorimetrically according to the Fiske and Subbarow method on 0.25 cc. of the solution.

B. Röntgenological examination

The left wrist of the infant was always examined, both in a frontal and in a lateral projection. Considering the debility of the infants, no repeated exposures of less satisfactory pictures

were allowed. The röntgenograms were taken in the obstetric department.

Afterwards each series of pictures was scrutinized by two röntgenologists, both with special experience of röntgen examination of infants, who independently of each other interpreted the pictures. In those cases where the interpretations differed, that one was accepted which deviated least from the normal finding.

C. Statistical methods

For the statistical analysis the material was, as already stated, divided into three groups according to birth weight, and these in their turn into age groups usually of 15 days each — in the cases in which 2 determinations on the same infant fell within such an age group, the mean of these two determinations was treated as 1 determination.

The statistical analysis was made according to methods described in Dahlberg's text-book (1940). The standard deviation is calculated according to the formula

$$\sigma = \pm \sqrt{\frac{\Sigma nd^2}{N-1}}$$

The standard error of the mean is calculated according to the formula

$$\varepsilon\,(M) = \pm \frac{\sigma}{\sqrt{N-1}}$$

The standard error of the difference between 2 means is calculated according to

$$\varepsilon^{(Diff.)} = \pm \sqrt{\varepsilon\,(M_1)^2 + \varepsilon\,(M_2)^2}$$

To be regarded as statistically significant a difference between 2 means had to amount to at least 3 times its standard error.

CHEMICAL DETERMINATIONS

I. Normal values

1. *Serum phosphatase.*

During the first week of life the mean value, as appears from Table 2, lies at about 7.5 phosphatase units. There is a rather great dispersion in the material. The lowest value observed during the first week of life is 2.8 units, the highest 14.8 units. —In the following week there seems to be a rise, for from and including the second half of the first month the values are at a new level and there remain little altered during the first six months. The mean is then about 16.5 units, and the difference between the mean of the neonatal period and the new mean is statistically significant. The dispersion is still great. The lowest observed value during this period is 6.4 units, the highest 29.4 units. Whether the latter value is to be regarded as normal is of course debatable. Statistically it lies just at the limit 3 times the standard deviation above the mean for the age-group 16—105 days. All told, values above 25 units have been obtained four times, i.e. values above twice the standard deviation, which is approximately what it should be if the material were normally distributed. These four infants showed clinically the same excellent condition as the rest of the material, except that the one with the highest phosphatase value was somewhat overfed and had less good home conditions than the majority of the others.

The reports of phosphatase values in healthy infants in the literature are rather scanty and expressed in several different kinds of units, which makes it difficult to compare them with

Age in days	Number of determinations	M	σ	ε(M)
1	22	8.355	±2.437	±0.532
2	22	8.014	±2.234	±0.487
3	23	7.426	±2.257	±0.481
1—3	*67*	*7.924*	*±2.378*	*±0.293*
4	19	7.021	±2.599	±0.613
5	22	6.945	±2.325	±0.507
6	21	7.424	±2.099	±0.469
4—6	*62*	*7.131*	*±2.306*	*±0.295*
1—6	**129**	**7.543**	**±2.367**	**±0.209**
16—45	*30*	*17.580*	*±4.449*	*±0.826*
46—75	*74*	*16.773*	*±4.190*	*±0.490*
76—105	*17*	*13.953*	*±3.266*	*±0.817*
16—105	**121**	**16.577**	**±4.251**	**±0.388**
106—180	23	*15.647*	*±3.296*	*±0.703*
16—180	**144**	**16.428**	**±4.117**	**±0.344**

Table 2. Serum phosphatase values in the normal material.

ours. Yet most investigators have observed that the values rise after the neonatal period. "The average value is low at birth, but rises abruptly to a maximum during the 1st month. This maximum level is maintained only a short time, though the average value remains at a comparatively high level until well into the 2nd year of life," say Stearns & Warweg (1933). Vermehren (1938), too, finds in his small series an indication of rise during the first months. Barnes & Munks (1940) found in a normal series of 390 observations that "the average serum phosphatase from birth to 3 days of age, has a value of 7.1 (Bodansky) units per 100 cc. It shows no peak during the 1st month but rather a sharp rise to 11.9 units during the first 2 months, a very slow continuance of this rise through the 4th and 5th months and then a gradual, slight decline through the rest of the year to a level of 11.5 units." — Our results thus

show quite good agreement with those obtained by earlier investigators.

In the small series of cow's milk fed infants, the serum phosphatase value is 14.511 ± 0.931 units. The standard deviation is ± 3.840 units. The difference between the breast-fed and the bottle-fed infants is not statistically significant or probable.

Vermehren (1938) has studied the *seasonal variation* of plasma phosphatase in a small series and found that the values were lower during the summer. The seasonal variation in our material appears from Table 3. For the neonatal period there is a slight, statistically insignificant, decline during the summer months. In the series of older infants the surprising fact is discovered that the values are highest during the autumn and lowest during the winter months: the difference between the periods September—November and December—February is statistically significant.

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.					29	17.076	±4.177	±0.789
July —Sept.					22	16.850	±4.739	±1.034
Aug. —Oct.					34	17.512	±4.397	±0.765
Sept. —Nov.					38	17.918	±4.123	±0.678
Oct. —Dec.					41	16.507	±4.724	±0.747
Nov. —Jan.	66	7.735	±2.458	±0.305	28	14.714	±4.213	±0.811
Dec. —Febr.	48	7.321	±2.369	±0.346	28	14.411	±3.994	±0.769
Jan. —March	47	7.864	±2.435	±0.359	24	16.563	±4.286	±0.894
Febr. —April	34	7.615	±2.571	±0.448	26	16.738	±4.271	±0.854
March—May	41	7.637	±2.547	±0.403	26	16.392	±4.124	±0.825
April —June	36	6.850	±1.957	±0.331	34	16.494	±3.426	±0.596
May —July	29	7.021	±1.861	±0.352	33	17.067	±3.836	±0.678

Table 3. Seasonal variation of serum phosphatase in the normal material.

2. *Serum inorganic phosphorus.*

From Table 4 it appears that the values for serum inorganic phosphorus during the whole period of observation are fairly closely distributed round the mean 6.23 mg %. The highest

observed value in the material is 7.85 mg %, the lowest 4.30 mg %. During the latter half of the first week there is a slight, but statistically significant decline which, however, is recovered in the second half of the first month.

Age in days	Number of determinations	M	σ	ε(M)
1	22	6.234	±0.761	±0.166
2	22	6.980	±0.760	±0.166
3	23	6.522	±0.852	±0.182
1—3	*67*	*6.578*	*±0.831*	*±0.102*
4	20	5.835	±0.575	±0.132
5	22	5.898	±0.671	±0.146
6	21	5.902	±0.729	±0.163
4—6	*63*	*5.879*	*±0.652*	*±0.083*
1—6	**130**	**6.239**	**±0.824**	**±0.073**
16—45	*30*	*6.292*	*±0.695*	*±0.129*
46—75	*74*	*6.252*	*±0.515*	*±0.060*
76—105	*17*	*5.924*	*±0.781*	*±0.195*
16—105	**121**	**6.216**	**±0.611**	**±0.056**
106—180	*23*	*6.248*	*±0.816*	*±0.174*
16—180	**144**	**6.221**	**±0.644**	**±0.054**
1—180	**274**	**6.230**	**±0.733**	**±0.044**

Table 4. Serum inorganic phosphorus in the normal material.

If we compare the values for the neonatal period with those earlier reported it is found that they agree quite well with the large series published by Todd et al. (1939). These investigators, on analysing 548 blood samples taken during the first three days of life found a mean of 6.08 mg %, and for 530 samples from the four following days a mean of 5.93 mg %. Denzer et al. (1939) found a mean of 7.11 mg % for 41 determinations from peripheral blood during the first week. In this material no decline could be shown during the end of the first week. Barnes & Munks (1940) found the mean 6.4 mg % in 45 determinations

from the first 3 days and from 23 determinations from 3—15 days the mean 6.8 mg %, which implies instead a rise but it is not statistically significant or probable. Bakwin (1937) also found in a small series often quoted, a rise during the neonatal period from 5.70 mg % during the first 2 days (only 7 determinations) to 6.14 mg % during the following 3, and 6.84 during the following 5 days. In the light of our observations of premature infants it is perhaps possible, at least in the case of Denzer, to associate the different behaviour of the phosphorus values in their material as distinguished from ours with the fact that in Denzer's material all the infants examined had had cow's milk to supplement the breast-feeding from the 3rd day.

Other investigators have reported considerably lower values for serum inorganic phosphorus during the neonatal period. At least to some extent this is probably because they have analysed the cord blood at birth. Todd et al. (1939) have collected from the literature 619 determinations of the cord blood and found a mean for it of 4.26 mg %, while 26 determinations after fontanel puncture collected in the same way gave a mean of 6.31 mg %. Further they have in 65 cases of their own compared the phosphorus values in cord blood and after fontanel puncture in the same infants and found in cord blood serum 5.54 mg % phosphorus, in fontanel blood serum from the first 3 days 6.20 and from the 4th to the 7th days 5.74 mg %. Denzer et al. (1939) made a similar comparison in their 41 cases and found that the value in the peripheral blood serum was on the average 1.05 mg % higher than in the cord blood serum

Normal values obtained from a fairly large series after the neonatal period seem to have been reported only by Barnes & Munks (1940). They found that after the second week the phosphorus value receded slightly with the rise of serum calcium at one month and then established a fairly constant though slightly falling value during the remainder of the year to an average of 5.8 mg at 12 months. For the age period 1/2—3 1/2 months one can calculate from their material a mean value for 91 determinations of 6.530 mg %. Bullock (1930) found a mean value of 5.47 mg % for 16 determinations on

infants between one and four months, and Stearns & Warweg (1933) found that for 39 artificially fed infants aged 2 weeks to 3 years "the inorganic phosphorus of serum rises steadily after birth (when it was 5.6 mg %) until the maximum value is reached at from 4 to 6 months."

Older authors generally gave lower normal values and these are quoted in most text-books. Thus Howland and Kramer (1923) gave the normal range as 4.5—6.0 mg % and A. F. Hess (1929) as 4.5—5.5 mg %. It is possible that the selection and feeding of their normal groups was not quite so careful as that of today's series. It is also possible that the difference is partly due to difference in method. Normal values obtained on whole blood instead of serum also lie at a considerably lower level (e. g. Vermehren 1938 and Josefsson 1941).

The serum inorganic phosphorus in our normal group fed cow's milk is 6.539 $\pm$ 0.112 mg %. The standard deviation is $\pm$ 0.463, i.e. somewhat less than in the normal group fed human milk. The difference between the cow's milk group and the human milk group is 2 1/2 times its standard error and thus may be considered statistically probable.

Some investigators have found greater or less *seasonal variations* for serum inorganic phosphorus. Thus, for example, Todd

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.					29	6.464	$\pm$0.725	$\pm$0.137
July —Sept.					22	6.386	$\pm$0.620	$\pm$0.135
Aug. —Oct.					34	6.309	$\pm$0.509	$\pm$0.089
Sept. —Nov.					38	6.168	$\pm$0.526	$\pm$0.086
Oct. —Dec.					41	6.126	$\pm$0.570	$\pm$0.090
Nov. —Jan.	66	6.168	$\pm$0.801	$\pm$0.099	28	6.002	$\pm$0.638	$\pm$0.123
Dec. —Febr.	49	6.116	$\pm$0.780	$\pm$0.112	28	6.109	$\pm$0.631	$\pm$0.121
Jan. —March	48	6.046	$\pm$0.811	$\pm$0.118	25	6.176	$\pm$0.437	$\pm$0.089
Febr. —April	35	6.204	$\pm$0.790	$\pm$0.135	26	6.213	$\pm$0.411	$\pm$0.082
March—May	41	6.311	$\pm$0.844	$\pm$0.133	26	6.123	$\pm$0.523	$\pm$0.105
April —June	36	6.447	$\pm$0.855	$\pm$0.145	33	6.244	$\pm$0.735	$\pm$0.130
May —July	29	6.444	$\pm$0.912	$\pm$0.172	33	6.303	$\pm$0.778	$\pm$0.138

Table 5. Seasonal variation of serum inorganic phosphorus in the normal material.

et al. (1939) found in new-born infants during the month March—May a mean of 6.10 mg %, as against 5.99 during the months June—August and September—November. The difference is small but was stated to be statistically significant. The seasonal variations in our material appear from Table 5. The variations in the different seasons are in no case statistically significant, but in some cases both in the neonatal material and in the older infants they may be considered statistically probable. The difference between the means for April—June and for January—March in the neonatal group is more than twice its standard error, and in the group of older infants the difference between June—August and November—January is more than 2 1/2 times the standard error of the difference. During the summer the values all round are somewhat higher than during the winter.

3. *Serum Calcium.*

Serum calcium has, as Table 6 shows, its mean for the first 3 days at about 9.73 mg %. The lowest observed value during this period is 8.1, the highest 11.3 mg %. After that a statistically significant rise occurs so that the values during the next three days are around a mean of 10.12 mg %, with an observed range between 7.4 and 12.2 mg %. The value 7.4 mg %, observed in a four-days-old infant, is more than 3 times the standard deviation from the mean and is probably not to be regarded as normal. The infant in question was, however, quite healthy all through the nursing period and showed normal values otherwise. If this one is excluded the lowest observed normal value during this time was 8.4 mg %.

During the course of the 2nd week of life a new statistically significant rise of the serum calcium value must take place, seeing that from and including the second half of the first month it is distributed around a new mean of 11.28 mg % which remains unchanged throughout the first half year of life. The lowest value observed during this period is 8.5, the highest 13.5 mg %. Also the value 8.5 mg % is outside the limit 3 times

Age in days	Number of determinations	M	σ	ε(M)
1	22	9.850	±0.754	±0.165
2	21	9.781	±0.657	±0.147
3	22	9.568	±0.611	±0.133
1—3	*65*	*9.732*	*±0.675*	*±0.084*
4	20	10.010	±0.909	±0.209
5	21	10.238	±0.704	±0.157
6	21	10.110	±0.726	±0.162
4—6	*62*	*10.121*	*±0.775*	*±0.099*
1—6	**127**	**9.922**	**±0.748**	**±0.067**
16—45	*30*	*11.170*	*±0.720*	*±0.133*
46—75	*74*	*11.239*	*±0.657*	*±0.077*
76—105	*17*	*11.300*	*±0.798*	*±0.200*
16—105	**121**	**11.231**	**±0.688**	**±0.063**
106—180	*23*	*11.548*	*±0.688*	*±0.147*
16—180	**144**	**11.281**	**±0.694**	**±0.058**

Table 6. Serum calcium in the normal material.

the standard deviation from the mean and can be pathological. The infant in question was a little overfed, but otherwise in very good condition, healthy and well-nursed and its other values were normal. Excepting this one the lowest observed value is 9.3 mg % which is within 3 times the standard deviation from the mean.

The small normal group fed cow's milk has a serum calcium of 11.367 ± 0.152 mg % with a standard deviation of ± 0.626 mg %, i.e. approximately the same as for the breast-fed infants.

Similarly, as regards the serum inorganic phosphorus, the values for serum calcium during the neonatal period show very good agreement with those published by Todd et al. (1939). They found for the first three days for 630 fontanel punctures a mean of 9.93 mg % (range 7.2—12.3 mg %) and for the next four days a mean of 10.45 mg % (7.5—13.9). — Denzer et al. (1939) found for 138 determinations a continuous rise from

10.56 mg % during the first and second days to 11.25 mg % during the 7th—10th days. Bakwin (1937) for 31 determinations from the 3rd—5th days of life found a mean of 9.76 mg % and for 23 determinations from the 6th—10th days 10.36 mg %. In cord blood higher values have been found than in the blood taken later during the neonatal period. Todd et al. (1939) and Denzer et al. (1939) have shown that the values in cord blood are on the average more than 1 mg % higher than in the blood sample taken during the first days of life from the same child, and Bakwin (1937), in a small series, found that serum calcium already 2—4 hours after birth can have fallen as far as to 2 mg % lower than in the cord blood at birth. The rise which takes place during the first weeks thus seems to be preceded by a fall during the first days of life. This fall will not appear in our material, but it may be possible to trace its effect in the inconsiderable fall which seems to take place from 1—3 days of age. This is, however, not statistically significant or probable.

From later infancy the investigations of Barnes & Munks (1940) are those chiefly available. They found that the serum calcium value after the 2nd week "rose to an average of 12.1 mg at the 1st month and were maintained rather consistently throughout the year". From these figures a mean can be calculated for the age-period 1/2—3 1/2 months of 11.875 mg % (87 determinations). — A. F. Hess (1933) from 17 determinations on infants found a value of 10.8 mg %, and Stearns & Warweg (1933) found a mean of 11.3 mg % for 54 determinations on bottle-fed infants of between 2 weeks and 3 years who had had small additions of vitamin D. Stearns (1939) states that after the neonatal period and during the whole of infancy the value generally lies about 1 mg % under that of the new-born, i.e. at about 10—12 mg %, with a rich supply of vitamin D near the upper limit, with a barely sufficient supply, near 10 mg %. Barnes (1945) gives as a normal level 10.8—12.0 mg %. Thus our values agree fairly well with those reported earlier.

Todd et al. (1939) found that in their material of new-born

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.					29	11.141	±0.795	±0.150
July —Sept.					22	11.427	±0.786	±0.172
Aug. —Oct.					34	11.474	±0.874	±0.152
Sept. —Nov.					38	11.374	±0.752	±0.124
Oct. —Dec.					41	11.256	±0.834	±0.132
Nov. —Jan.	63	10.006	±0.838	±0.106	28	11.211	±0.738	±0.142
Dec. —Febr.	46	10.093	±0.562	±0.084	28	11.079	±0.750	±0.144
Jan. —March	48	9.958	±0.539	±0.079	24	11.150	±0.478	±0.100
Febr. —April	35	9.914	±0.562	±0.096	26	11.169	±0.462	±0.092
March—May	41	9.844	±0.601	±0.095	26	11.285	±0.313	±0.063
April —June	36	9.792	±0.650	±0.110	31	11.155	±0.527	±0.096
May —July	29	9.748	±0.736	±0.139	31	11.052	±0.531	±0.097

Table 7. Seasonal variation of serum calcium in the normal material.

infants there existed a slight but definitely increased value for calcium during the autumn and winter. They attributed this "possibly to the prolonged effect of the summer's sun, with increased mobilization of calcium in the blood stream, or possibly to the maternal consumption of more fresh vegetables during the spring and summer, with resulting higher calcium values in the succeeding seasons." From Table 7 it is clear that in our new-born group also the mean is highest during the winter months and falls as the summer approaches. The difference between November—January and May—July can possibly be regarded as statistically probable, as it exceeds 2 but not 2 1/2 times its standard error. — For the older infants the values lie highest during the autumn and lowest during the spring and early summer, but the differences are here in no case statistically significant or probable.

4. *Serum total protein.* According to Peters and van Slyke (1935) one cannot interpret the values for serum calcium without having at the same time determined both the serum protein and serum inorganic phosphorus, or knowing that those are normal. For this reason a rough determination of serum total protein has been included in this investigation.

Age in days	Number of determinations	M	σ	s(M)
1	22	6.300	±0.473	±0.103
2	21	6.481	±0.596	±0.133
1—2	*43*	*6.388*	*±0.554*	*±0.082*
3	23	5.970	±0.460	±0.098
4	19	6.105	±0.644	±0.152
5	21	6.014	±0.400	±0.089
6	20	6.140	±0.475	±0.109
3—6	*83*	*6.053*	*±0.489*	*±0.054*
1—6	**126**	**6.167**	**±0.529**	**±0.047**
16—45	*30*	*5.570*	*±0.381*	*±0.071*
46—75	*74*	*5.655*	*±0.384*	*±0.045*
76—105	*17*	*5.824*	*±0.435*	*±0.109*
16—105	**121**	**5.658**	**±0.394**	**±0.036**
106—180	*23*	*5.752*	*±0.329*	*±0.070*
16—180	**144**	**5.673**	**±0.383**	**±0.032**

Table 8. Serum protein in the normal material.

As appears from Table 8 the protein, during the first two days of life, lies at about 6.4 % (the highest observed value 8.1, the lowest 5.4 %). After that there is a statistically significant decline to about 6.05 % during the rest of the first week (7.7—4.5 %) and this seems to continue through the second week to a level round about 5.65 % which is maintained fairly constant during the rest of the first 6 months. The highest observed value in the older infants is 6.8 %, the lowest 4.8 %. The values during the neonatal period vary, as appears, quite considerably, and the two highest observed values exceed 3 times the standard deviation from the mean in the groups concerned. The values for serum calcium in the cases where the serum protein values differ more considerably from the mean show nothing worthy of attention.

The little normal group fed cow's milk had about the same serum protein value as the human milk group, or 5.661 ± 0.092 %, with a standard deviation of 0.381 %.

Denzer et al. (1939) examined the serum protein content in 79 cases during the first 10 days of life and found means for the different days which varied between 6.03 and 5.89 %, with a range for the individual values between 5.10 and 7.53 %. They used the refractometric method. "No correlation could be found between the steady level of protein and the fluctuations in the calcium level".—Hickmans et al. (1943) determined the plasma-protein content in 180 normal full-term infants up to 40 weeks of age with a micro-Kjeldahl method. During the first two weeks the values lay between 4 and 7 %, in most cases under 6 %. In the older infants the range was 4.7—7.4 %, but in the children over 10 weeks old most of the values lay in the upper half of this range, i.e. between 6.0 and 7.4 %.—A. F. Hess (1933) found the mean for serum protein in 17 healthy infants to be 6.4 %.

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.					29	5.755	±0.478	±0.090
July —Sept.					22	5.473	±0.290	±0.063
Aug. —Oct.					34	5.403	±0.292	±0.051
Sept. —Nov.					38	5.439	±0.272	±0.045
Oct. —Dec.					41	5.466	±0.265	±0.042
Nov. —Jan.	62	5.989	±0.491	±0.063	28	5.514	±0.219	±0.042
Dec. —Febr.	45	5.996	±0.567	±0.085	27	5.567	±0.257	±0.050
Jan. —March	48	6.085	±0.472	±0.069	24	5.667	±0.290	±0.060
Febr. —April	35	6.306	±0.539	±0.092	26	5.742	±0.307	±0.061
March—May	41	6.334	±0.456	±0.072	26	5.965	±0.361	±0.072
April —June	36	6.361	±0.555	±0.094	34	6.000	±0.430	±0.075
May —July	29	6.383	±0.487	±0.092	33	5.973	±0.438	±0.077

Table 9. Seasonal variation of serum protein in the normal material.

I have not found any communication in the literature regarding the *seasonal variations* for serum protein in infants. From Table 9 it appears that in our material the values in the new-born show a striking, statistically significant rise from the winter months to the spring and early summer. For the older infants the values are lowest during the autumn and then rise

steadily to the highest values which are reached during the latter part of the spring and early summer. Here too the differences between the autumn and the spring values are statistically significant. The correlation with the values for serum calcium is obvious from this arrangement: when the mean for serum calcium is highest, the serum protein is lowest, and vice versa. The seasonal variations of serum calcium, are however, not statistically significant in this material

Summary.

The following normal values were obtained with the methods used on serum from healthy breast-fed infants during the first six months, who after the neonatal period received an ample supply of vitamin D: (round figures for the means together with the limits $\pm\sigma$, i.e. the limits within which two-thirds of all the normal values observed ought to fall).

Serum phosphatase: First week of life 7.5 units (5.0—10.0 units). After the second week of life 16.5 units (12—20 units).

Seasonal variation: After the second week of life on an average higher values in summer than in winter.

Serum inorganic phosphorus: 6.25 mg % (5.5—7.0 mg %). During the end of the first week somewhat lower values.

Seasonal variation: Probably somewhat higher values in the summer than in winter.

Serum calcium: First three days of life 9.75 mg % (9.0—10.5 mg %).

Following three days 10.1 mg % (9.25—11.0 mg %).

After second week of life 11.25 mg % (10.5—12.0 mg %).

Seasonal variation: In the newborn probably higher values in winter than in summer.

Serum total protein: First two days of life 6.4 %o (5.9—6.9 %o).

The rest of the first week 6.0 %o (5.5—6.5 %o).

After second week of life 5.7 %o (5.3—6.1 %o).

Seasonal variation: highest values in the spring and early summer, lowest in the autumn and winter.

II. Premature infants fed human milk without any supplement

To this group have been reckoned such infants who since birth and before this determination have been fed exclusively human milk without any extra supply of either vitamin D or minerals. In the highest age groups have also been included a few determinations on infants who had earlier had a supplement of cows milk or of some mineral preparation, but who during at least the last 25 days before the determination were fed exclusively human milk. 6 determinations were included from 4 infants who had been given an amino acid preparation in addition to the human milk. The values obtained from these infants fall well within the variation range for the infants fed exclusively human milk. 6 infants are included to whom citric acid (and 4 % sugar) had been added to the human milk (in the same way as in preparing citric acid milk from cow's milk) in order to test the antirachitic effect of citric acid formulated by Hamilton (1933) and others. The 11 determinations carried out upon these 6 infants gave values which fall wholly within the variation range of the other infants fed exclusively human milk, and are therefore included in this group.

All in all this group consists of 213 determinations performed on 108 infants. Of these, however, 64 infants belonged to the group only at the first examination. As previously mentioned the serum inorganic phosphorus determinations carried out at the beginning of the investigation were rejected, so that the number of phosphorus determinations included is less. The

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	ε(Diff.)
1—3	*39*	*12.438*	*± 4.181*	*±0.678*	*4.512*	*±0.699*
4—6	*36*	*11.983*	*± 3.392*	*±0.573*	*4.853*	*±0.606*
7—15	*34*	*17.659*	*± 7.263*	*±1.264*		
16—30	50	30.444	±12.013	±1.716		
31—45	31	35.513	±14.510	±2.649		
16—45	*81*	*32.384*	*±13.078*	*±1.462*	*14.792*	*±1.683*
46—75	*19*	*32.974*	*±16.165*	*±3.810*	*16.201*	*±3.915*
76—105	*4*	*29.750*			*15.797*	
16—105	**104**	**32.390**	**±13.630**	**±1.343**	**15.813**	**±1.398**
16—105: Birth weight:						
1010—1500	15	43.673	±19.617	±5.243	27.096	±5.257
1510—1750	32	33.053	±14.632	±2.629	16.476	±2.658
1760—2000	57	29.049	± 9.629	±1.287	12.472	±1.323

Table 10. Serum phosphatase in premature infants fed human milk without any supplement.

number of serum protein determinations is also less because the serum protein was not determined at the beginning of the investigation.

1. *Serum phosphatase.* — From Table 10 it is seen that the phosphatase values in this group during the neonatal period are on an average 1 and 1/2 times as high as in the normal material. The difference is statistically significant. The dispersion is rather great. The highest observed value during the first week of life is 20.8, the lowest 5.0 units. — During the second week of life the values lie significantly higher than during the first, and during the following months they remain round about a mean of 32 units, or double as high as in the normal. The dispersion is very great. For the smallest premature infants, with a birth weight between 1 000 and 1 500 gms, the mean lies considerably higher than for the other birth weight groups, and the difference between them and the highest birth-

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.	26	11.958	±3.951	±0.790	30	28.437	± 8.702	±1.616
July —Sept.	16	14.094	±3.165	±0.817	21	30.924	±12.552	±2.807
Aug. —Oct.	21	13.557	±3.743	±0.837	16	34.219	±15.066	±3.890
Sept. —Nov.	19	13.289	±4.525	±1.066	18	37.233	±13.560	±3.289
Oct. —Dec.	18	12.856	±4.580	±1.111	18	32.650	±12.054	±2.924
Nov. —Jan.	18	11.772	±5.027	±1.219	21	26.776	±11.655	±2.606
Dec. —Febr.	17	11.288	±3.997	±0.999	23	25.878	±11.773	±2.510
Jan. —March	19	11.205	±3.807	±0.897	30	28.837	±13.332	±2.476
Febr. —April	15	11.940	±2.837	±0.758	26	31.612	±12.900	±2.580
March—May	11	11.545	±3.133	±0.991	18	33.056	±12.258	±2.973
April —June	20	10.625	±3.616	±0.830	20	30.565	± 8.671	±1.989
May —July	19	10.874	±3.779	±0.891	26	30.069	± 7.896	±1.579

Table 11. Seasonal variation of serum phosphatase in premature infants fed human milk without any supplement.

weight group, 1 760—2 000 gms, is statistically probable. The highest observed values in the three birth weight groups of this age period are 83.2, 64.4 and 53.0 units, the lowest 20.4, 10.0 and 11.4 units.

Table 11 shows the seasonal variations of the phosphatase values. In the neonatal group there is a rise during the end of the summer and the autumn, and the lowest means are found in the spring and early summer. The difference between July—September and April—June periods is almost exactly 3 times its standard error and thus almost significant statistically. In the higher age group the lowest values are found during the winter, and then there is a continuous rise to the maximum during the autumn. The difference between September— November and December—February periods is more then $2^1/_2$ times its standard error and can be termed statistically probable. — Most noticeable is the close congruence between the seasonal variation in this group and in the normal material. In the higher age group the congruence is almost absolute with the maximum and minimum at exactly the same season. For the new-born the minimum value lies in both cases in the period April—June,

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	ε(Diff.)
1—3	*25*	*6.104*	*±1.161*	*±0.237*	*—0.474*	*±0.257*
4—6	*34*	*5.181*	*±1.145*	*±0.199*	*—0.693*	*±0.214*
7—15	*28*	*4.516*	*±0.962*	*±0.185*		
16—30	44	4.442	±1.242	±0.150		
31—45	23	4.726	±1.372	±0.293		
16—45	*67*	*4.540*	*±1.284*	*±0.153*	*—1.777*	*±0.202*
46—75	*13*	*4.277*	*±1.135*	*±0.328*	*—1.880*	*±0.279*
76—105	*3*	*4.217*			*—1.707*	
16—105	**83**	**4.487**	**±1.236**	**±0.136**	**—1.729**	**±0.147**
16—105: Birth weight:						
1010—1500	15	3.717	±0.931	±0.248	—2.499	±0.255
1510—1750	24	4.646	±1.119	±0.248	—1.570	±0.255
1760—2000	44	4.662	±1.288	±0.196	—1.554	±0.202

Table 12. Serum inorganic phosphorus in premature infants fed human milk without any supplement.

while the maximum value in the premature infants lies in a season for which no normal values are available.

In analysing the seasonal variations the determinations on infants in the lowest birth weight group (1 010—1 500 gms) have been omitted, because they deviate so much from the other values obtained and are so unequally distributed over the seasons that they might upset the comparison.

2. *Serum inorganic phosphorus.* — From Table 12 it is clear that serum inorganic phosphorus during the first days of this group lies very nearly at the same level as in the normal material. The difference is statistically insignificant. The highest observed value during the first days is 8.70, the lowest 3.10 mg %. During the latter half of the first week the value in this group decreases more than in the normal material, and the difference between them is now statistically significant. The dispersion in the premature group is now also significantly greater than in the

normal material. The highest observed value during the latter part of the first week is 6.85, the lowest 3.25 mg %. — After that the difference increases still more. While in the normal material the mean now returns to about the original value, in this group it continues to decline, and lies for the rest of the observation period at a level somewhat under 4.5 mg %. The dispersion is great and the values observed range between 8.00 and 2.10 mg %. The mean for the values from the lowest birth weight group is lower than from the others and the difference is statistically probable.

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.	15	5.790	±1.034	±0.276	21	4.840	±1.105	±0.247
July —Sept.	10	5.400	±1.045	±0.348	12	4.729	±1.218	±0.367
Aug. —Oct.	17	5.350	±0.900	±0.225	8	4.537	±1.177	±0.445
Sept. —Nov.	19	5.524	±1.305	±0.308	14	4.414	±1.303	±0.361
Oct. —Dec.	18	5.667	±1.436	±0.348	17	4.318	±1.334	±0.334
Nov. —Jan.	16	5.406	±1.468	±0.379	21	4.633	±1.486	±0.332
Dec. —Febr.	13	5.073	±1.077	±0.311	20	4.810	±1.591	±0.365
Jan. —March	15	4.947	±1.165	±0.311	23	4.902	±1.330	±0.284
Febr. —April	13	5.731	±1.469	±0.424	18	4.497	±1.280	±0.310
March—May	10	5.815	±1.584	±0.526	13	4.385	±0.765	±0.221
April —June	14	6.121	±1.337	±0.371	16	4.609	±1.075	±0.278
May —July	11	5.814	±1.148	±0.363	21	4.862	±1.030	±0.230

Table. 13. Seasonal variation of serum inorganic phosphorus in premature infants fed human milk without any supplement.

Table 13 shows the s e a s o n a l v a r i a t i o n of the serum inorganic phosphorus in this group (the determinations for the lowest birth weight class being omitted). It should be noted that the values vary during the neonatal period in this group in exactly the same way as in the normal material. The values lie lowest during the winter, highest during the spring and summer. The difference between the period January—March and April—June can possibly be regarded as statistically probable as it is more than double its standard error. — For the higher age group on the other hand no seasonal variation can be demonstrated in this group.

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	ε(Diff.)
1—3	*39*	*8.659*	*±1.560*	*±0.253*	*—1.073*	*±0.266*
4—6	*34*	*9.435*	*±1.316*	*±0.229*	*—0.686*	*±0.249*
7—15	*33*	*9.679*	*±1.382*	*±0.244*		
16—30	49	9.645	±1.173	±0.169		
31—45	31	9.884	±0.857	±0.156		
16—45	*80*	*9.737*	*±1.083*	*±0.122*	*—1.433*	*±0.182*
46—75	*19*	*10.016*	*±0.880*	*±0.207*	*—1.223*	*±0.221*
76—105	*4*	*10.025*			*—1.275*	
16—105	**103**	**9.800**	**±1.037**	**±0.122**	**—1.431**	**±0.103**
16—105: Birth weight:						
1010—1500	15	9.600	±1.371	±0.366	—1.631	±0.371
1510—1750	32	9.959	±1.213	±0.218	—1.272	±0.228
1760—2000	56	9.762	±0.831	±0.112	—1.469	±0.130

Table 14. Serum calcium in premature infants fed human milk without any supplement.

3. *Serum Calcium.* — From Table 14 it is seen that the serum calcium values during the whole period of observation are significantly lower than in the normal material. During the first 3 days of life the mean lies about 1 mg % lower than in the normal material. After that there is a rise quite definite and statistically significant during the second half of the first week, and this rise seems to continue somewhat during the period of observation, but it was not possible to demonstrate any statistically significant difference between the age groups later on. The highest and lowest observed values during the first three days were 13.8 and 5.2 mg %, during the next three days 12.2 and 7.0 mg %, and during the later age period 12.4 and 6.3 mg %. No symptoms of tetany were observed in those cases showing extremely low serum calcium values. — The means for the different birth weight classes show no very clear difference, but the dispersion is greater in the lower classes. The

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.	24	8.837	±1.780	±0.371	29	9.724	±1.001	±0.189
July —Sept.	15	9.800	±1.474	±0.394	21	10.076	±1.025	±0.229
Aug. —Oct.	19	9.647	±1.438	±0.339	15	10.213	±1.102	±0.294
Sept. —Nov.	17	9.071	±1.285	±0.321	18	9.350	±1.222	±0.296
Oct. —Dec.	18	8.856	±1.155	±0.280	17	9.218	±1.221	±0.305
Nov. —Jan.	18	9.444	±1.658	±0.402	21	9.495	±1.131	±0.253
Dec. —Febr.	16	9.606	±1.604	±0.414	24	10.092	±0.880	±0.183
Jan. —March	18	9.367	±1.513	±0.367	31	10.187	±0.771	±0.141
Febr. —April	14	8.493	±0.879	±0.244	27	10.181	±0.797	±0.156
March —May	11	8.573	±1.110	±0.351	17	10.171	±0.586	±0.147
April —June	20	8.295	±1.600	±0.367	19	9.542	±0.717	±0.169
May —July	19	8.479	±1.617	±0.381	24	9.508	±0.816	±0.170

Table 15. Seasonal variation of serum calcium in premature infants fed human milk without any supplement.

dispersion, as is seen, is greatest in the lowest age groups; the extreme values occur almost exclusively during the first month.

Table 15 shows the seasonal variation of serum calcium. Here, too, the determinations on infants in the lowest birth weight group have been omitted. During the neonatal period the values are lowest during the spring and early summer, highest during the autumn. The difference between the periods July—September and April—June is almost statistically significant. The minimum coincides quite well with the minimum for the normal values during the neonatal period. The annual curve for the higher age groups shows a more peculiar course with two maxima, at the beginning of autumn and the beginning of winter, and two minima, at the beginning of winter and the end of spring. Both declines are statistically probable.

4. *Serum total protein.* — Table 16 shows that the values in this group lie on the average somewhat lower than in the normal material, and the difference in each age group is statistically significant. As in the normal material the mean declines

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	ε(Diff.)
1—3	*9*	*5.467*	*±0.531*	*±0.188*	*—0.775*	*±0.197*
4—6	*26*	*5.246*	*±0.519*	*±0.104*	*—0.839*	*±0.122*
7—15	*19*	*5.416*	*±0.416*	*±0.098*		
16—30	32	5.134	±0.501	±0.090		
31—45	14	4.886	±0.582	±0.161		
16—45	*46*	*5.059*	*±0.527*	*±0.079*	*—0.511*	*±0.105*
46—75	*10*	*4.920*	*±0.390*	*±0.130*	*—0.735*	*±0.170*
76—105	*2*	*5.250*			*—0.574*	
16—105	**58**	**5.014**	**±0.502**	**±0.067**	**—0.644**	**±0.071**
16—105: Birth weight:						
1010—1500	12	4.783	±0.456	±0.137	—0.875	±0.141
1510—1750	19	5.111	±0.363	±0.086	—0.547	±0.089
1760—2000	27	5.107	±0.582	±0.114	—0.551	±0.118

Table 16. Serum protein in premature infants fed human milk without any supplement.

as the age increases, and the decline from the first days of life to the age group 16—105 days is statistically significant. The highest and lowest values observed during the neonatal period are 6.3 and 4.3 %, during the rest of the observation period 6.0 and 4.0 %. The infants in the lowest birth weight class have during the age period 16—105 days lower serum protein than the heavier premature infants: this difference can possibly be termed statistically probable as it is greater than twice its standard error.

The seasonal variation appears from Table 17. No significant seasonal variation during the neonatal period can be demonstrated. During the latter part of the observation period, on the other hand, there is a definite seasonal variation with high values during the spring and summer, and low ones in the autumn, just as in the normal material. The difference

Season	Infants 1—6 days old				Infants 16—105 days old			
	No. of determ.	M	σ	ε(M)	No. of determ.	M	σ	ε(M)
June —Aug.	15	5.413	±0.574	±0.153	21	5.252	±0.436	±0.097
July —Sept.	10	5.260	±0.477	±0.159	11	5.209	±0.373	±0.118
Aug. —Oct.	13	5.185	±0.486	±0.140	7	5.171	±0.414	±0.169
Sept. —Nov.	9	5.178	±0.567	±0.200	6	4.800	±0.654	±0.292
Oct. —Dec.	7	5.214	±0.624	±0.255	5	4.380	±0.423	±0.212
Nov. —Jan.	6	5.267	±0.491	±0.220	9	4.489	±0.366	±0.129
Dec. —Febr.	6	5.367	±0.421	±0.188	12	4.833	±0.455	±0.137
Jan. —March	7	5.429	±0.428	±0.175	14	5.029	±0.425	±0.118
Febr. —April	6	5.150	±0.409	±0.183	10	5.330	±0.381	±0.127
March—May	5	5.120	±0.448	±0.224	7	5.414	±0.333	±0.136
April —June	11	5.318	±0.613	±0.194	16	5.338	±0.485	±0.125
May —July	10	5.570	±0.629	±0.210	20	5.255	±0.447	±0.103

Table 17. Seasonal variation of serum protein in premature infants fed human milk without any supplement.

between the periods March—May and October—December is statistically significant.

Summary. — *Premature infants with a birth weight not above 2 000 gms, and fed human milk without any supplement of either vitamin D or minerals, show the following main deviations from the normal serum values:*

Serum phosphatase: significantly higher values, in the new-born 1 1/2 times, in the following months twice the normal values. Great dispersion. The premature infants with the lowest birth weight have the highest serum phosphatase values. *Seasonal variation* quite congruent with that of normal values, both in the new-born and later.

Serum inorganic phosphorus: at birth at the same level as the normal values: then declines, and from and including the second week lies at about 4.5 mg %: i. e. nearly 2 mg % lower than the normal values. Great dispersion. For the premature infants in the lowest birth weight class the mean lies below 4 mg %. *Seasonal variation* in the new-born congruent with that of the normal values, later no seasonal variation.

Serum calcium: During the first days about 1 mg % lower than the normal values. Then rises like the normal values but somewhat more slowly and after the second week lies at about 1 1/2 mg % lower than the normal values. Great dispersion. *Seasonal variation* in the new-born probably congruent with that of the normal values, lowest in the spring, highest in the autumn. During the following months two maxima, at the beginning of autumn and the beginning of spring, and two minima, at the beginning of winter and at the end of spring.

Serum total protein: At birth about 1 % under the normal value, later somewhat less difference. Seasonal variation after the neonatal period congruent with that of the normal values.

III. Premature infants fed human milk + vitamin D

The Vitamin D was supplied either in the form of D-vimin (Astra) in a dose containing 5 000, 7 500 or 10 000 I.U. vitamin D_3 daily or as Ultranol fortior (Ferrosan) in a single massive dose containing 500 000 I.U. vitamin D_2. For the statistical analysis only those determinations have been included which were carried out after the infant had been treated with D-vimin for at least the previous 25 days, or at least 25 days earlier had been given the massive dose of Ultranol fortior. (25 days was made the limit because rather many determinations were made about 28 days after the beginning of the treatment.) All the infants included had been fed exclusively human milk for the last 25 days before the determination: some few of them had, before that time for a short period, received an addition of cow's milk or some mineral salt preparation.

The material thus selected comprises 60 determinations on 32 infants: 29 determinations on 17 infants given 5 000 I.U. of D-vimin daily: 4 determinations on 4 children given 7 500 I.U. of D-vimin daily: 10 determinations on 7 infants given 10 000 I.U. of D-vimin daily: 17 determinations on 9 children given a single massive dose of Ultranol fortior.

Serum phosphatase. — As appears from Table 18 this group shows in general the same values as the group of premature infants fed human milk without vitamin D. Yet the values show a greater tendency than in the latter group to approach the normal values with increasing age, and the mean for the age

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
31—45	*17*	*31.371*	*±14.553*	*±3.638*	*13.791*[1]	*±3.731*	*—4.142*	*±4.500*
46—75	*19*	*30.374*	*±17.661*	*±4.162*	*13.601*	*±4.191*	*—2.600*	*±5.642*
76—105	*12*	*25.242*	*±12.644*	*±3.812*	*11.289*	*±3.900*	*—4.508*	
31—105	**48**	**29.444**	**±15.214**	**±2.219**	**12.867**[2]	**±2.253**	**—4.749**	**±3.024**
Birth weight:								
1510—1750	19	37.832	±20.505	±4.833	21.255[2]	±4.849	2.576	±6.219
1760—2000	29	23.948	± 6.924	±1.308	7.371[2]	±1.365	—5.538	±2.274
106—180	12	*20.475*	*±10.437*	*±3.147*	*4.828*	*±3.225*		

Table 18. Serum phosphatase in premature infants fed human milk + vitamin D.

group 106—180 days is not significantly higher than the normal mean. When dividing the values according to different birth weights it will be seen, too, that the higher birth weight group has values a little lower than the corresponding group fed human milk without vitamin D: the difference can perhaps be termed statistically probable, as it exceeds twice its standard error. The standard deviation is also less in this birth weight group than in the corresponding group given no vitamin D, and the difference between the standard deviations also exceeds twice its standard error. In the lower birth weight group, on the other hand, there is no difference between the mean for the groups with and without vitamin D, and the standard deviation for those given vitamin D is rather greater than for those given no vitamin D: this difference however is not significant or probable. — The extreme values observed in the lower birth weight group between 31 and 105 days of life are 16.8 and 89.6 units, in the higher birth weight group, 12.2 and 43.2 units. Between 106—180 days of life, the extreme values observed are 9.2 and 49.6 units. — No clear difference between the values

[1] Normal value for 16— 45 days.

[2] Normal value for 16—105 days.

Season	No. of determ.	M	σ	ε(M)	Season	No. of determ.	M	σ	ε(M)
June—Aug.	10	25.640	± 6.743	±2.248	Dec. —Febr.	16	37.681	±20.990	±5.42
July —Sept.	12	28.967	±10.483	±3.160	Jan. —March	8	31.288	±14.026	±5.30
Aug. —Oct.	10	25.510	±12.186	±4.062	Febr. —April	9	22.400	± 5.361	±1.89
Sept.—Nov.	11	30.355	±15.739	±4.976	March—May	13	21.454	± 6.422	±1.85
Oct. —Dec.	17	35.429	±21.424	±5.356	April —June	13	20.915	± 6.695	±1.93
Nov.—Jan.	18	39.439	±20.119	±4.880	May —July	13	23.500	± 7.776	±2.24

Table 19. Sesonal variation of serum phosphatase in premature infants 31—105 days o fed human milk + vitamin D.

after the different doses of vitamin D can be deduced from this small material.

Table 19 shows that in this group also there is a marked seasonal variation. During the spring and summer the values are low with no great dispersion, during the autumn and winter they are as much as double as high and show an extraordinarily great dispersion. Compared with the normal material and the group of premature infants fed human milk without vitamin D there is in this series a displacement, so that the maximum and minimum occur 2 and 3—4 months later respectively, giving rise to the curious result that the maximum coincides with the minimum in these groups and vice versa. The seasonal groups, however, are small and therefore no great importance can be ascribed to the variation.

2. *Serum inorganic phosphorus.* — Table 20 shows that the phosphorus value in premature infants given a large dose of vitamin D is the same as in premature infants given no vitamin D. The rise with increasing age which can be traced in the Table is quite insignificant. Dividing the values according to birth weight it is found, however, that, as was the case with the phosphatase values, the higher birth weight group has a little higher mean than the corresponding group given no vitamin D: the difference can possibly be termed statistically probable as it exceeds twice its standard error. Also just as

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	Diff. from premat. inf. fed only hum. milk
31—45	*16*	*4.934*	*±1.043*	*±0.269*	*—1.358*[1] *±0.298*	*0.308 ±0.397*
46—75	*17*	*4.785*	*±0.700*	*±0.175*	*—1.467 ±0.187*	*0.508 ±0.372*
76—105	*10*	*5.360*	*±1.125*	*±0.375*	*—0.564 ±0.423*	*1.143*
31—105	**43**	**4.974**	**±0.944**	**±0.146**	**—1.242**[2] **±0.156**	**0.437 ±0.249**
Birth weight:						
1510—1750	19	4.766	±1.064	±0.251	—1.450[2] ±0.257	0.229 ±0.322
1760—2000	24	5.140	±0.825	±0.172	—1.076[2] ±0.181	0.603 ±0.265
106—180	*11*	*5.186*	*±0.696*	*±0.220*	*—1.062 ±0.226*	

Table 20. Serum inorganic phosphorus in premature infants fed human milk + vitamin D.

with phosphatase the dispersion in the higher birth weight group is smaller than in the corresponding group given no vitamin D; and this difference too can possibly be termed statistically probable as it exceeds twice its standard error. In the lower birth weight group, the mean and the dispersion are the same as in the corresponding group given no vitamin D. The highest and the lowest observed values during the age period 31—105 days are in the lower birth weight group 7.85 and 3.45 mg %, in the higher 6.45 and 3.05 mg %. During the age period 106—180 days the highest and the lowest observed values were 5.90 and 3.65 mg %. No clear difference between the values after the different doses of vitamin D could be discerned.

From Table 21 it appears that the seasonal variation coincides with that in the group of premature infants without vitamin D and also in the normal material. The values are low during the winter and high during the summer.

[1] Normal value for 16— 45 days.
[2] Normal value for 16—105 days.

Season	No. of determ.	M	σ	ε(M)	Season	No. of determ.	M	σ	ε(M)
June —Aug.	8	5.750	±1.306	±0.494	Dec. —Febr.	15	4.780	±0.873	±0.233
July —Sept.	8	5.181	±1.502	±0.568	Jan. —March	8	4.612	±0.525	±0.198
Aug. —Oct.	5	4.830	±1.013	±0.507	Febr. —April	9	4.944	±0.555	±0.196
Sept. —Nov.	8	4.525	±0.912	±0.345	March—May	9	5.000	±0.601	±0.213
Oct. —Dec.	15	4.807	±0.986	±0.263	April —June	12	5.288	±0.559	±0.169
Nov. —Jan.	17	4.656	±0.883	±0.221	May —July	12	5.508	±1.112	±0.333

Table 21. Seasonal variation of serum inorganic phosphorus in premature infants 31—10[illegible] days old fed human milk + vitamin D.

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
31—45	*17*	*10.741*	*±0.563*	*±0.171*	*—0.429*[1]	*±0.221*	*0.857*	*±0.235*
46—75	*17*	*10.582*	*±0.847*	*±0.212*	*—0.657*	*±0.226*	*0.566*	*±0.297*
76—105	9	*10.800*	*±0.796*	*±0.281*	*—0.500*	*±0.345*	*0.775*	
31—105	**43**	**10.691**	**±0.706**	**±0.109**	**—0.540**[2]	**±0.126**	**0.750**	**±0.164**
Birth weight:								
1510—1750	17	10.600	±0.587	±0.147	—0.631[2]	±0.161	0.641	±0.272
1760—2000	26	10.750	±0.787	±0.155	—0.481[2]	±0.170	0.988	±0.192
106—180	*8*	*11.263*	*±0.788*	*±0.284*	*—0.285*	*±0.321*		

Table 22. Serum calcium in premature infants fed human milk + vitamin D.

3. *Serum calcium.* — As appears from Table 22 the value for serum calcium in this group is considerably higher than in premature infants fed human milk but given no vitamin D. It is not, however, so high as that of the normal material. The differences from both the groups are statistically significant. The dispersion is the same as in the normal material. The highest observed value in the age-group 31—105 days was 12.4, the lowest 8.7 mg %. As in the group of premature infants fed

[1] Normal values for 16— 45 days.

[2] Normal values for 16—105 days.

Season	No. of determ.	M	σ	ε(M)	Season	No. of determ.	M	σ	ε(M)
June —Aug.	10	10.730	±0.596	±0.199	Dec. —Febr.	15	10.787	±0.576	±0.154
July —Sept.	11	10.991	±0.799	±0.253	Jan. —March	7	10.514	±1.053	±0.421
Aug. —Oct.	7	11.200	±0.844	±0.345	Febr. —April	6	10.200	±1.039	±0.465
Sept. —Nov.	9	10.978	±0.807	±0.285	March—May	9	10.200	±0.899	±0.318
Oct. —Dec.	15	10.820	±0.526	±0.141	April —June	10	10.290	±0.522	±0.174
Nov. —Jan.	18	10.761	±0.559	±0.136	May —July	12	10.533	±0.615	±0.185

Table 23. Seasonal variation of serum calcium in premature infants 31—105 days old fed human milk + vitamin D.

human milk without vitamin D, there is no difference between the birth weight groups nor can any difference be demonstrated between the serum calcium values after the different doses given of vitamin D.

The seasonal variation appears from Table 23. The values are high in the autumn and low in the winter. The maximum in the period August—October coincides with the maximum for the groups previously analysed. The number of determinations in the different seasons is small, but the difference between the periods July—December and January—June can possibly be termed statistically probable as it exceeds twice its standard error.

4. *Serum total protein.* — For the age group 31—105 days the mean of 19 determinations is 5.042 ± 0.097 %, i. e. the same as in the group of premature infants fed human milk but given no vitamin D. The standard deviation is 0.412 %. The highest value observed in this group was 6.2 %, the lowest 4.4 %. For the age group 106—180 days the mean of 6 determinations is 5.233 %. — As in the previous groups the lowest values are found in the autumn. The number of determinations, however, is too small to allow a more exact statistical analysis.

S u m m a r y. — *Premature infants with a birth weight not above 2 000 gms and fed human milk plus an ample supply of vitamin D for at least 25 days show in their serum values the following main differences from premature infants fed human milk but given no vitamin D, and from the normal:*

Serum phosphatase: no significant difference from premature infants given no vitamin D: yet, in the premature infants of the highest birth weight class investigated, there are possibly values a little lower in the group given vitamin D: similarly, in premature infants given vitamin D there is possibly a tendency towards lower values with increasing age. *Seasonal variation:* high values in the autumn and winter, low in the spring and the summer: the maximum and minimum occur some months later than in the normal material and in premature infants given no vitamin D.

Serum inorganic phosphorus: no significant difference from premature infants fed human milk without vitamin D: yet, those of the highest birth weight class can possibly be said to have values a little higher when given vitamin D. — *The seasonal variation* coincides with that in the normal material and in premature infants fed human milk without vitamin D.

Serum calcium: significantly higher values than in premature infants fed human milk but given no vitamin D, but still *significantly lower than in normal infants. — Seasonal variation:* high values in the autumn, low in the spring.

Serum total protein: no difference from premature infants fed human milk but given no vitamin D.

IV. Premature infants given cow's milk but no vitamin D

The infants included in this group were given a certain amount of a cow's milk formula for some time before the actual examination as a supplement to the human milk or, in some instances, as the sole food. At the beginning of the investigation a half milk formula with 5 % sugar and a thin oat gruel was given: Later citric acid whole milk with 4 % sugar was given. As the values obtained after half milk and after citric acid milk never differ significantly from each other the two series are combined. The amounts of cow's milk given daily have varied between 100 and 440 cc: no significant difference between the values after giving more and less cow's milk has been found: nor any difference between the values obtained after different periods of cow's milk feeding: these periods have varied between 3 days and nearly 3 months.

This group comprises 34 determinations performed on 20 infants: 14 determinations on 5 infants given half milk: and 20 determinations on 15 infants given citric acid whole milk.

1. *Serum phosphatase.* — In the age period 16—105 days the values in this group, as will be seen from Table 24, are much lower than in the group of premature infants fed only human milk, but still higher than in the normal material: the differences from both these groups are statistically significant. The tendency towards higher values with increasing age as suggested in the table is, of course, quite insignificant, considering the small number of determinations in each age-group. The

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	Diff. from premat. inf. fed only hum. milk
4—6	*1*	*12.600*			*5.469*	*0.617*
7—15	*1*	*13.400*				*— 4.259*
16—30	4	16.800				—13.644
31—45	9	25.356				—10.157
16—45	*13*	*22.723*	*± 9.471*	*±2.734*	*5.143 ±2.856*	*— 9.661 ±3.100*
46—75	*10*	*24.750*	*± 8.532*	*±2.844*	*7.977 ±2.886*	*— 8.224 ±4.763*
76—105	*7*	*23.400*			*9.447*	*—6.350*
16—105	**30**	**23.497**	**± 9.602**	**±1.783**	**6.920 ±1.823**	**— 8.893 ±2.232**
16—105 Birth weight:						
1010—1500	3	30.867			14.290	—12.806
1510—1750	9	26.478			9.901	— 6.575
1760—2000	18	20.778	± 8.280	±2.008	4.201 ±2.045	— 8.271 ±2.385
106—180	2	*32.000*			*16.353*	
16—180	**32**	**24.028**	**±10.280**	**±1.846**	**7.600 ±1.878**	

Table 24. Serum phosphatase in premature infants given cow's milk but no vitamin D.

same is true of the higher value in the lower birth weight groups. No significant seasonal variations can be observed. The dispersion is considerably higher than the normal: the lowest value observed was 10.2, the highest, 47.2.

2. *Serum inorganic phosphorus.* — Table 25 shows that the values for serum inorganic phosphorus in this group in the age-period 16—105 days are much higher than in premature infants fed only human milk, and the difference is statistically significant. Yet it does not quite reach the values of the normal group; the difference from the normal can possibly be termed statistically probable as it is twice its standard error. The standard deviation is somewhat smaller, though not significantly so, than that of the premature infants fed only human milk, and

Age in days	Number of determinations	M	σ	s(M)	Diff. from normal	Diff. from premat. inf. fed only hum. milk
7—15	*1*	*7.400*				*2.884*
16—30	1	5.050				0.608
31—45	9	5.900				1.174
16—45	*10*	*5.815*	*±0.638*	*±0.213*	*—0.477 ±0.249*	*1.275 ±0.265*
46—75	*10*	*5.525*	*±0.804*	*±0.268*	*—0.727 ±0.276*	*1.248 ±0.424*
76—105	*7*	*6.279*			*0.355*	*2.062*
16—105	**27**	**5.828**	**±0.931**	**±0.183**	**—0.388 ±0.190**	**1.341 ±0.235**
16—105: Birth weight:						
1010—1500	3	6.150			—0.066	2.433
1510—1750	9	5.922			—0.294	1.276
1760—2000	15	5.707	±0.764	±0.204	—0.509 ±0.212	1.045 ±0.288
106—180	*2*	*5.625*			*—0.623*	
16—180	**29**	**5.814**	**±0.899**	**±0.170**	**—0.407 ±0.179**	

Table 25. Serum inorganic phosphorus in premature infants given cow's milk but no vitamin D.

somewhat greater than in the normal group: here the difference can be termed statistically probable as it is 2 1/2 times its standard error; the lowest value observed was 4.00 and the highest, 7.60 mg %. — No significant differences between the groups of different ages or different birth weights can be discerned and no significant seasonal variations.

3. *Serum calcium.* — As will appear from Table 26 the supply of cow's milk has not altered in any way the serum calcium values in premature infants. The difference from premature infants fed only human milk is quite insignificant. The standard deviation is also quite the same as in the group of premature infants fed only human milk. The lowest value observed was 8.0 and the highest, 11.4 mg %. No differences between the dif-

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
4—6	1	*9.500*			*—0.621*		*0.065*	
7—15	1	*9.000*					*—0.679*	
16—30	4	9.150					—0.495	
31—45	9	9.900					0.016	
16—45	*13*	*9.669*	*±1.042*	*±0.301*	*—1.501*	*±0.330*	*—0.068*	*±0.318*
46—75	*10*	*9.660*	*±1.388*	*±0.463*	*—1.579*	*±0.469*	*—0.356*	*±0.507*
76—105	*6*	*9.600*			*—1.700*		*—0.425*	
16—105	**29**	**9.652**	**±1.093**	**±0.207**	**—1.579**	**±0.217**	**—0.148**	**±0.241**
16—105: Birth weight:								
1010—1500	2	9.200			—2.031		—0.400	
1510—1750	9	9.589			—1.642		—0.370	
1760—2000	18	9.733	±1.021	±0.248	—1.498	±0.257	—0.029	±0.271
106—180	*1*	*9.500*			*—2.048*			
16—180	**30**	**9.647**	**±1.074**	**±0.199**	**—1.634**	**±0.207**		

Table 26. Serum calcium in premature infants given cow's milk but no vitamin D.

ferent age groups or birth weight groups, and no seasonal variation can be discerned from the low figures available.

4. *Serum total protein.* — The mean for the 17 determinations of serum protein in the age period 16—105 days is 5.418 ± 0.161 %. This is a little higher than in premature infants fed only human milk and the difference is perhaps statistically probable as it exceeds twice its standard error. The standard deviation is 0.632 % and the lowest value observed is 4.5, the highest 6.7 %.

S u m m a r y. *Premature infants with a birth weight not above 2 000 gm and given a supply of cow's milk but no vitamin D, are characterized by the following peculiarities in their serum values:*

Serum phosphatase. — Significantly higher than normal, lower than in premature infants fed only human milk.

Serum inorganic phosphorus. — Significantly higher than in premature infants fed only human milk, yet not quite as high as normal.

Serum calcium. — Quite the same as in premature infants fed only human milk.

Serum total protein. — Possibly a little higher than in premature infants fed only human milk.

V. Premature infants given cow's milk + vitamin D

The infants included in this group have been given cow's milk just as the preceding group, and also vitamin D, like the group reported in chapter III. The vitamin D supply was given for at least 25 days immediately before the determination.

The group comprises 60 determinations carried out on 38 infants: 11 determinations on infants given half milk; of these, 2 determinations after giving 5 000 I.U. of D-vimin daily; 3 determinations after giving 10 000 I.U. of D-vimin daily; 6 determinations after a single massive dose of 500 000 I.U. of ultranol fortior. 49 determinations were carried out on infants given citric acid whole milk; of these, 8 determinations after giving 5 000 I.U. of D-vimin daily; 1 determination after 7 500 and 30 determinations after 10 000 I.U. of D-vimin daily, and 10 determinations after a single massive dose of ultranol fortior. No significant differences could be obtained between the values after giving half milk and citric acid whole milk, or between the values after the different doses of vitamin D. The amounts of cow's milk given daily have varied between 100 and 700 cc.; significant difference could be obtained after giving the different amounts of cow's milk.

1. *Serum phosphatase.* — As Table 27 shows, the serum phosphatase values in this group are the same as in the normal group and significantly lower than in the other groups of premature infants, even the group given cow's milk but no vitamin D. The dispersion too is the same as in the normal

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
16—45	*5*	*17.080*			*—0.500*		*—15.304*	
46—75	*15*	*16.353*	*±5.384*	*±1.439*	*—0.426*	*±1.659*	*—16.621*	*±4.073*
76—105	*16*	*16.681*	*±4.403*	*±1.137*	*2.728*	*±1.400*	*—13.069*	
16—105	**36**	**16.600**	**±4.899**	**±0.828**	**0.023**	**±0.915**	**—15.790**	**± 1.578**
16—105: Birth weight:								
1010—1500	1	11.600			—4.977		—32.063	
1510—1750	15	19.160	±5.057	±1.351	2.583	±1.406	—13.893	±2.956
1760—2000	20	14.930	±4.089	±0.938	—1.647	±1.015	—14.119	±1.592
106—180	*24*	*17.029*	*±4.854*	*±1.012*	*1.382*	*±1.232*		
16—180	**60**	**16.774**	**±4.828**	**±0.629**	**0.346**	**±0.717**		

Table 27. Serum phosphatase in premature infants given cow's milk + vitamin D.

group and significantly smaller than in the other premature groups. The highest value observed was 27.6 units, the smallest 9.3 units. The mean for the birth weight group 1510—1750 gm. of the age group 16—105 days is somewhat higher than for the corresponding higher birth weight group, and the difference may be statistically probable as it exceeds $2\,^1/_2$ times its standard error. No difference was obtained between the different age groups, and no significant seasonal variation was observed.

2. *Serum inorganic phosphorus.* — Table 28 shows that the serum inorganic phosphorus in this group, just like the serum phosphatase, is almost exactly the same as in the normal group and significantly higher than the groups of premature infants fed human milk with or without vitamin D. The difference from the group given cow's milk but no vitamin D, however, is not statistically significant or probable. The standard deviation from the mean is the same as in premature infants given cow's milk but no vitamin D and in those given human milk + vitamin D and greater than that of the normal group; this difference

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
16—45	*5*	*5.720*			*—0.572*		*1.180*	
46—75	*15*	*6.420*	*±0.832*	*±0.222*	*0.168*	*±0.230*	*2.143*	*±0.394*
76—105	*15*	*6.223*	*±1.148*	*±0.307*	*0.299*	*±0.361*	*2.006*	
16—105	**35**	**6.236**	**±0.912**	**±0.156**	**0.020**	**±0.164**	**1.749**	**±0.205**
16—105: Birth weight:								
1010—1500	1	5.500			—0.716		1.783	
1510—1750	14	6.461	±0.983	±0.273	0.245	±0.279	1.815	±0.370
1760—2000	20	6.115	±0.919	±0.211	—0.101	±0.219	1.453	±0.293
106—180	*22*	*6.543*	*±0.803*	*±0.175*	*0.295*	*±0.232*		
16—180	**57**	**6.354**	**±0.857**	**±0.115**	**0.133**	**±0.126**		

Table 28. Serum inorganic phosphorus in premature infants given cow's milk + vitamin D.

can be termed statistically probable as it exceeds 2 1/2 times its standard error. On the other hand, the standard deviation is less than in premature infants fed human milk without vitamin D and this difference can possibly be termed statistically probable as it exceeds twice its standard error. The lowest value observed in this group is 4.50 mg %, the highest, 8.20 mg %. No significant difference can be obtained between the different birth weight groups or age groups, and no significant seasonal variation can be deduced from the figures obtained.

3. *Serum calcium.* — Table 29 shows that the serum calcium value is a little lower in this group than in the normal, and much higher than in the group of premature infants fed human milk without any supplement, and in the group of premature infants given cow's milk but no vitamin D. The differences from these groups are statistically significant. On the other hand it can be termed the same as in the premature infants given human milk+vitamin D; the difference between these

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
16—45	*5*	*10.600*			*—0.570*		*0.863*	
46—75	*15*	*10.953*	*±0.491*	*±0.131*	*—0.286*	*±0.152*	*0.937*	*±0.245*
76—105	*13*	*10.723*	*±1.012*	*±0.292*	*—0.577*	*±0.354*	*0.698*	
16—105	**33**	**10.809**	**±0.724**	**±0.128**	**—0.422**	**±0.141**	**1.009**	**±0.176**
16—105: Birth weight:								
1010—1500	1	9.100			—2.131		—0.500	
1510—1750	15	10.933	±0.707	±0.189	—0.298	±0.200	0.974	±0.290
1760—2000	18	10.706	±0.769	±0.187	—0.525	±0.197	0.944	±0.219
106—180	*22*	*10.736*	*±0.756*	*±0.165*	*—0.812*	*±0.221*		
16—180	**55**	**10.780**	**±0.724**	**±0.099**	**—0.501**	**±0.114**		

Table 29. Serum calcium in premature infants given cow's milk + vitamin D.

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
16—45	*5*	*5.480*			*—0.090*		*0.421*	
46—75	*13*	*4.969*	*±0.356*	*±0.103*	*—0.686*	*±0.110*	*0.049*	*±0.167*
76—105	*8*	*5.250*			*—0.574*		*0*	
16—105	**26**	**5.154**	**±0.396**	**±0.079**	**—0.504**	**±0.084**	**0.140**	**±0.100**
16—105: Birth weight:								
1010—1500	1	4.500			—1.158		—0.283	
1510—1750	13	5.077	±0.355	±0.102	—0.581	±0.105	—0.034	±0.130
1760—2000	13	5.231	±0.438	±0.126	—0.427	±0.130	0.124	±0.170
106—180	*11*	*5.600*	*±0.512*	*±0.162*	*—0.152*	*±0.176*		
16—180	**37**	**5.286**	**±0.465**	**±0.078**	**—0.387**	**±0.084**		

Table 30. Serum protein in premature infants given cow's milk + vitamin D.

groups is not statistically significant or probable. The standard deviation from the mean can be termed the same in this group, in the normal group, and in the group of premature infants given human milk + vitamin D; it is smaller than in the group of premature infants given no vitamin D but the difference is not significant or probable. The lowest value observed in this group was 8.2 mg %, and the highest, 11.8 mg %. No significant difference could be obtained between the different birth weight groups or age groups, and no significant seasonal variation.

4. *Serum total protein.* — Table 30 shows that the serum protein in this group is the same as in premature infants fed only human milk and significantly lower than normal. No significant differences can be observed between the different age groups or birth weight groups, and no seasonal variation is confirmed. The highest value observed is 6.6 %, the lowest 4.2 %.

S u m m a r y. — *Premature infants with a birth weight not above 2 000 gm and given a supply of cow's milk + a large dose of vitamin D, are distinguished by the following main peculiarities of their serum values:*

Serum phosphatase. — The same as normal value, and significantly lower than all the other groups of premature infants investigated; yet probably a little higher in the lower birth weight group than in the higher. The dispersion, too, is the same as in the normal group.

Serum inorganic phosphorus. — The same as normal value, and significantly higher than in the groups of premature infants fed human milk. The dispersion is greater than in the normal group.

Serum calcium. — The same as in the group of premature infants fed human milk + vitamin D; a little lower than in the normal group and much higher than in the groups of premature infants given no vitamin D. The dispersion is the same as in the normal group and in the premature infants fed human milk + vitamin D.

Serum total protein. — The same as in premature infants fed only human milk and significantly lower than the normal.

VI. Premature infants given different supplies of mineral preparations

In order to discover whether the improved serum values obtained by giving cow's milk supplies to premature infants could also be obtained by supplementing the human milk feeding with other sources of mineral salts, some assays have been made with a series of different mineral preparations.

Secondary calcium phosphate, $CaHPO_4$, was given to 3 infants for 2—4 weeks in a dose of 0.155 gm per 100 cc human milk, by which the calcium and phosphorus content of the human milk was increased to that of half cow's milk. The 3 determinations carried out after this treatment, at the age of 31—60 days, gave for serum phosphatase values between 29.8 and 56.4 units; for serum inorganic phosphorus between 3.70 and 4.20 mg %; and for serum calcium between 8.7 and 10.8 mg %. The serum protein varied between 4.2 and 4.9 %. In no case, had there been an obvious improvement of the values since the preceding determination.

Ash of a milk powder preparation, solved in lactic acid and then neutralized with sodium hydroxide, was given to 3 infants for 3—7 days in a dose containing about 0.125 gm calcium and 0.075 gm phosphorus per 100 cc. human milk, by which the human milk was brought up to the calcium and phosphorus content of cow's milk. Two of the infants had for a month been given a daily dose of 5 000 units of *D-vimin.* The assays were carried out at the age of 31—60 days. — The serum values after giving the ash were for serum phosphatase

Age in days	Number of determinations	M	σ	s(M)	Diff. from normal		Diff. from premat. inf. fed only hum. milk	
7—15	*1*	*12.000*					*— 5.659*	
16—45	*13*	*26.831*	*±11.758*	*±3.241*	*9.251*	*±3.344*	*— 5.553*	*±3.556*
46—75	*7*	*27.100*			*10.327*		*— 5.874*	
76—105	*1*	*47.200*			*33.247*		*17.450*	
16—105	**21**	**27.890**	**±13.271**	**±2.968**	**11.313**	**±2.993**	**—4.500**	**±3.258**
16—105: Birth weight:								
1010—1500	4	49.725			33.148		6.052	
1510—1750	9	23.911			7.334		— 9.142	
1760—2000	8	21.450			4.873		— 7.599	

Table 31. Serum phosphatase in premature infants given human milk + glucofos.

between 38.8 and 45.6 phosphatase units; for serum inorganic phosphorus between 6.20 and 7.00 mg %; and for serum calcium between 10.4 and 11.8 mg %. For serum protein the values were between 4.3 and 5.1 %. — In each case there was a large increase in serum inorganic phosphorus, from 3.70—4.60 mg % by 1.60 to 3.30 mg % in a week. In one case there was also a definite increase in serum calcium but the other values were unchanged. In two cases the treatment was accompanied by a certain loss in weight.

A *cow's milk whey powder,* containing 0.050 gm phosphorus and 0.054 gm calcium per gm, was given to one infant for 4 weeks, in a dose of 1 gm per 100 cc human milk. All values remained at about the same level as before treatment.

Finally, 10 infants were for varying periods given a *calcium hexosediphosphate* preparation, "Glucofos" (Astra), containing 17 % calcium and 13.1 % phosphorus. Of this the infants were given 0.220 or 0.660 gm per 100 gm human milk, by which the calcium content of the milk was increased by 37.4 or 112.2

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	Diff. from premat. inf. fed only hum. milk
7—15	*1*	*9.700*				*5.184*
16—45	*13*	*5.500*	*±1.057*	*±0.305*	*—0.792 ±0.332*	*0.960 ±0.467*
46—75	*7*	*5.300*			*—0.952*	*1.023*
76—105	*1*	*2.800*			*—3.124*	*—1.417*
16—105	**21**	**5.305**	**±1.088**	**±0.243**	**—0.911 ±0.249**	**0.818 ±0.277**
16—105: Birth weight:						
1010—1500	4	4.762			—1.454	1.045
1510—1750	9	5.422			—0.794	0.776
1760—2000	8	5.444			—0.772	0.782

Table 32. Serum inorganic phosphorus in premature infants given human milk + glucofos.

mg %, and the phosphorus content by 28.6 or 85.8 mg %, i.e. to the content of half or whole cow's milk, respectively. The preparation was given for between 7 and 49 days before the determinations, 4 determinations being made after giving the small dose, and 18 after the large dose.

As will be seen from table 31, the serum phosphatase is not significantly lower than in premature infants given human milk without any supplement; it is on the other hand, however, not significantly higher than in premature infants given cow's milk. The standard deviation is the same as in premature infants given human milk without any supplement.

The serum inorganic phosphorus, as will be seen from table 32, is higher than in premature infants fed only human milk; this difference is nearly statistically significant. It is on the other hand significantly lower than normal, and also lower than in premature infants given cow's milk, though not significantly. The standard deviation is a little greater than in premature infants given cow's milk, and a little smaller than in those fed only human milk, though the dif-

Age in days	Number of determinations	M	σ	ε(M)	Diff. from normal	Diff. from premat. inf. fed only hum. milk
7—15	*1*	*9.800*				*0.121*
16—45	*13*	*9.369*	*±0.671*	*±0.193*	*—1.801 ±0.235*	*—0.368 ±0.228*
46—75	*7*	*9.743*			*—1.496*	*—0.273*
76—105	*1*	*7.700*			*—3.609*	*—2.325*
16—105	**21**	**9.414**	**±0.881**	**±0.197**	**—1.817 ±0.207**	**—0.386 ±0.232**
16—105: Birth weight:						
1010—1500	4	8.800			—2.431	—0.800
1510—1750	9	9.650			—1.581	—0.309
1760—2000	8	9.412			—1.819	—0.350

Table 33. Serum calcium in premature infants given human milk + glucofos.

ference is not significant or probable in either case. — The extremely high value in the age group 7—15 days, obtained after giving the salt for 7 days, is remarkable. It is the highest value obtained since the phosphorus method was corrected.

Table 33 shows that the serum calcium is about the same as in premature infants given human milk or cow's milk without vitamin D. The standard deviation, too, is about the same as in these groups.

The serum protein in this group is 5.567 ± 0.100 % with a standard deviation of 0.447 %. This is about the same as in the normal group and as in premature infants given cow's milk but no vitamin D, and significantly higher than in premature infants given human milk without supplement.

In two cases determinations have been made on infants given the large dose of glucofos, who had received 10 000 I.U. *D-vimin* daily for at least 25 days. The serum phosphatase values in these cases were 9.0 and 21.0 I.U. respectively, the serum inorganic phosphorus values 5.60 and 5.95 mg %, and the serum calcium values 8.5 and 10.4 mg % respectively. The serum protein values were 6.2 and 5.3 % respectively.

Summary. — The addition of secondary calcium phosphate or of the minerals of cow's milk whey to human milk in a dose which increases its calcium and phosphorus content to about that of half cow's milk, does not seem to improve the serum values at all.

The addition of cow's milk ash to human milk, so as to increase the calcium and phosphorus content to that of cow's milk, can increase the serum inorganic phosphorus value considerably in a short time. However, this preparation does not seem to be borne quite well by the infants.

The addition of calcium hexosediphosphate in a dose which increases the calcium and phosphorus content of the human milk to that of half or whole cow's milk acts in about the same manner as does the feeding of cow's milk. However, the effect of the salt seems not to be so pronounced as that of cow's milk feeding. — The addition of calcium hexosediphosphate + vitamin D should be checked by further assays.

VII. A comparison of the serum values in the different groups

In *the first days of life,* premature infants have about the same serum inorganic phosphorus as full term infants. The serum phosphatase, on the other hand, is higher than normal, and the serum calcium already lower. The serum protein is also lower than normal.

In *the following months* the serum values vary with the food, at least to a certain extent. This is illustrated, for the age period 16—105 days, in figs 1—3. The columns represent the means of the groups ± the standard deviations, i.e. the range within which about two-thirds of the individual values may be expected.

1. *Serum phosphatase.* — When *only human milk* is given, the phosphatase values are much higher than normal; the dispersion of the values is also very great. The *addition of vitamin D* seems to make no significant difference in this; however, when the groups are divided into different birth weight classes, it appears that the higher class (fig. 2) given vitamin D has a significantly lower mean, and a slighter dispersion than the lower birth weight class given vitamin D, probably also higher than the higher birth weight class given no vitamin D. In the heavier premature infants, accordingly, vitamin D seems to have a certain effect on the phosphatase values. — *The addition of cow's milk,* on the other hand, gives significantly lower serum

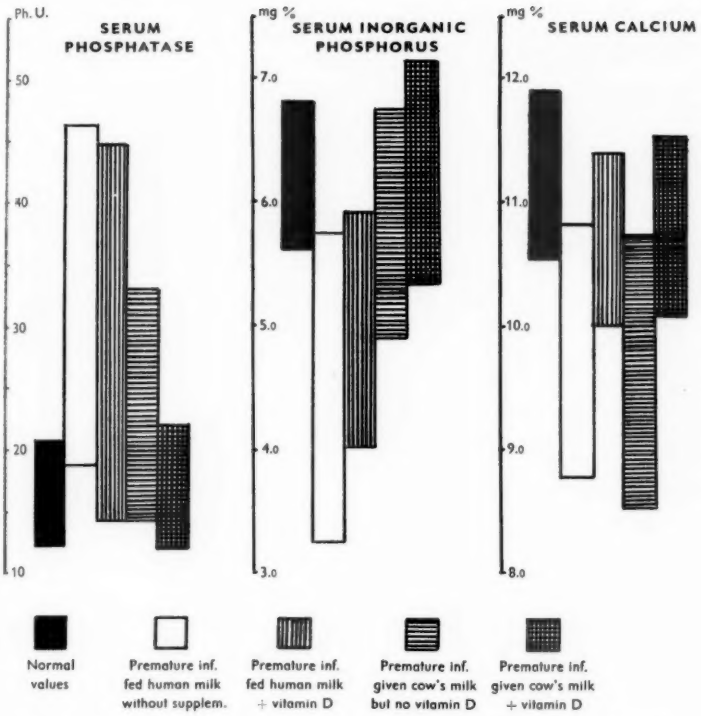


Fig. 1. Premature infants, birth weight 1010—2000 gm:serum values at 16—105 days of age after different feedings.

phosphatase values than does human milk alone, but still significantly higher than normal; the standard deviation too, is significantly greater than normal. This is true for both the birth weight groups. Finally, *the addition of cow's milk and vitamin D,* gives quite normal values for serum phosphatase, with quite normal dispersion.

2. *Serum inorganic phosphorus.* — *Human milk alone* gives phosphorus values much below normal, with very great dispersion. The *addition of vitamin D* to the

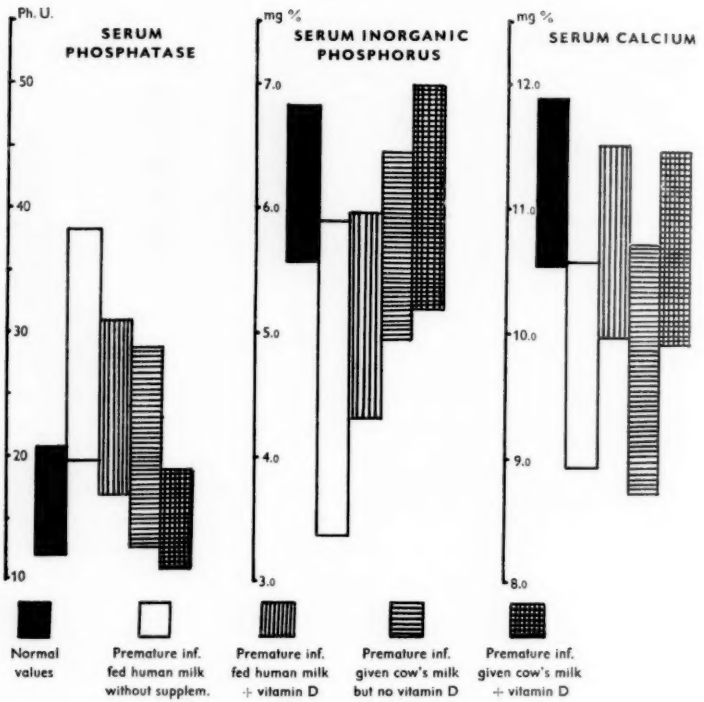


Fig. 2. Premature infants, birth weight 1510—1750: serum values at 16—105 days of age after different feedings.

human milk makes no significant difference; in the higher birth weight class there is possibly a slight increase. — The *addition of cow's milk,* increases the phosphorus value much more, to almost the normal level, and *the addition of both cow's milk and vitamin D* makes the values quite normal, though the dispersion is still a little greater than the normal. The difference between the values after cow's milk + vitamin D, and after cow's milk only, is still insignificant, so it cannot be

On Pages 74 and 75, the titles of the Figures 2 and 3 have been exchanged in error.

Thus, Fig. 2 should represent premature infants, birth weight 1760—2000, and Fig. 3 premature infants, birth weight 1510—2000.

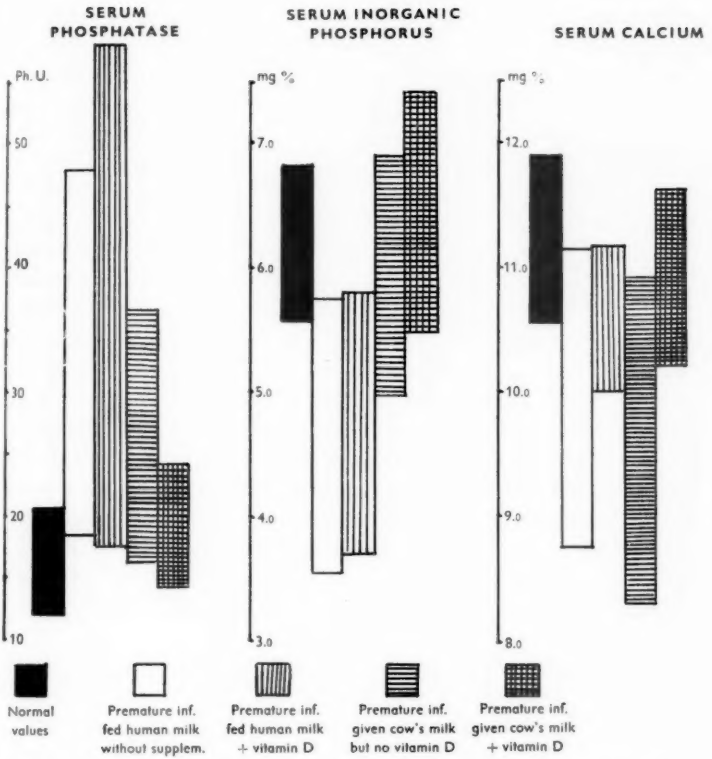


Fig. 3. Premature infants, birth weight 1760—2000: serum values at 16—105 days of age after different feedings.

said to prove any effect of vitamin D apart from that of the cow's milk.

3. *Serum calcium.* — As with phosphorus, on human milk alone the values sink to a level much below normal, with a great dispersion. Unlike the phosphorus, however, *the addition of cow's milk* does not improve this at all, whereas *the addition of vitamin D* results in

significantly better calcium values with a standard deviation as slight as in the normal group. *The addition of cow's milk and vitamin D* gives no better results than does the addition of cow's milk alone.

4. *Serum total protein.* — The serum protein is significantly lower than the normal and at the same level in all premature groups, with the curious exceptions of the one given cow's milk but no vitamin D and of that given "glucofos" but no vitamin D. However, these groups are quite small, and the protein method is a rough one, so it is wise not to draw any conclusions from the higher values thus obtained.

The main effect of vitamin D on the serum values, thus, is *to increase the serum calcium.* When given in addition to cow's milk it also significantly decreases the serum phosphatase, but when given with human milk only, this is not the case in the smaller premature infants, but probably to a certain extent in the heavier premature infants. In the latter also vitamin D can possibly slightly increase the serum inorganic phosphorus.

The addition of cow's milk causes a considerable increase in serum inorganic phosphorus, to almost the normal value, *and a considerable decrease in serum phosphatase;* in the latter case, the effect is further improved by giving vitamin D. The addition of cow's milk, on the other hand, has no effect at all on the serum calcium values.

To a certain degree, an effect similar to that resulting from giving cow's milk may be obtainable by *adding some mineral preparation,* such as calcium hexosediphosphate, to the human milk. This, however, demands further investigation.

RÖNTGENOLOGICAL EXAMINATIONS

I. Occurence of different röntgen signs

The röntgenological examinations are listed in Table 34. Where the same infant had been examined more than once in one age period, all examinations in the same age period were reckoned as one, provided they had to be included in the same group; if not, they were reckoned as one in each group. The total material thus consists of 443 examinations. Of these, 223, or half the number, were found to be *normal;* of the rest, in 156 examinations both the röntgenologists consulted interpreted the changes as *sure signs of rickets* in an active or healing stage. In 45 examinations the sole change was a ribbon-shaped, narrow zone of decreased density to the röntgen rays, lying some mm. proximally to the epiphyse line: this seems to be the same picture that Lindblom (1939) termed *"metaphyseal decalcification"*. Finally, in 19 cases, the sole change observed was one or more thin, straight lines of increased density running parallel to the end of the shaft: clearly the *"transverse lines"* of Eliot, Souther and Park (1927).

The changes interpreted as *signs of active or healing rickets* are termed, according to Eliot and Park (1945), *"cupping", "spreading", "spur formation", "fringing", "general decalcification", calcification at the epiphyse line,* and *"periosteal encasements"* of the shafts.

As appears from the table, the "metaphyseal decalcification" is practically limited to the first month of life, whereas the "transverse lines" have only occurred after the first month of life. "Signs of rickets" may occur, occasionally, as early as in

Age in days	Normal pictures Birth weight			Metaphyseal decalcif. Birth weight			Transverse lines Birth weight			Signs of rickets (active or healed) Birth weight			Total
	1010—1500	1510—1750	1760—2000	1010—1500	1510—1750	1760—2000	1010—1500	1510—1750	1760—2000	1010—1500	1510—1750	1760—2000	
1—3	—	12	23	—	2	1	—	—	—	—	—	—	**38**
4—6	—	7	10	—	1	2	—	—	—	—	—	—	**20**
7—15	1	5	13	2	4	3	—	—	—	1	—	—	**29**
16—30	1	15	33	3	8	11	—	—	—	—	3	5	**79**
31—45	—	8	25	—	3	3	—	1	3	5	13	16	**77**
46—60	—	6	16	—	2	—	—	3	2	6	15	12	**62**
61—75	—	4	12	—	—	—	—	2	2	7	13	9	**49**
76—105	—	6	10	—	—	—	—	2	2	6	12	8	**46**
106—180	—	6	10	—	—	—	—	1	1	5	12	8	**43**
1—180	**2**	**69**	**152**	**5**	**20**	**20**	—	**9**	**10**	**30**	**68**	**58**	**443**

Table 34. Distribution of röntgen examinations according to birth weight and age of the infants when examined.

the first month of life (the earliest observation in this study is from the 14th day of life) but from the second month onwards they become common: in the lowest birth-weight group all examinations after the first month of life show signs of rickets, in the middle group about two-thirds of the examinations, and in the highest birth-weight group, about two-fifths of the examinations.

What is the first sign of rickets in premature infants? What happens in the skeleton when different supplements are given to the food? What are the serum values at the first appearance of rachitic changes? — In order to study these problems those röntgen examinations were selected where a certain röntgen sign first appeared in an infant who had been previously examined at least within the last three weeks (usually about two weeks), or who was not more than one month old at the examination. This selection gave a certain guarantee that the sign had not been present for more than two or three weeks at the examination.

1. *Metaphyseal decalcification* occurred in 35 infants who fulfilled these demands. As appears from Table 35, it seldom starts after the first month of life. This may be the reason why nearly all the infants were such as had been given only human milk, as the decalcification was often observed already at the first examination, before starting the assay. Of the 6 infants given vitamin D, only one had taken it for more than 25 days, the rest for 15—19 days. One infant had been given cow's milk for 14 days, and two glucofos for 10 and 32 days.

In no case had the metaphyseal decalcification been preceded by any other röntgen changes, and only in two cases starting after the first month of life, did other changes (cortical spurs, fringing, spreading) appear at the same time as the decalcification. On the other hand, in nearly all the cases röntgen signs of rickets were observed at subsequent examinations: of the 7 infants in whom no other changes ever appeared, one died soon after the first examination; of the remaining six, examin-

Age in days	Total no.	Birth weight			Feeding					Further röntgen signs			
		1010—1500	1510—1750	1760—2000	human milk		cow's milk		mineral suppl.	preceding	coincident	subsequent	none
					no D	+ D	no D	+ D					
1—3	2	—	2	—	2	—	—	—	—	—	—	2	—
4—6	3	—	1	2	3	—	—	—	—	—	—	2	1
7—15	7	2	3	2	7	—	—	—	—	—	—	6	1
16—30	18	3	7	8	12			—	—	—	—	9	3
						5				—	—	4	1
							1			—	—	—	1
31—45	4	—	2	2	2		—	—		—	1	2	—
						1				—	1	1	—
									1	—	—	1	—
46—60	1	—	1	—	—	—	—	—	1	—	—	1	—
1—60	**35**	**5**	**16**	**14**	**26**	**6**	**1**	**—**	**2**	**—**	**2**	**28**	**7**

Table 35. First appearance of "metaphyseal decalcification".

ations some weeks later revealed quite normal pictures; one of them, however, at a control at the age of 3 months, showed marked rachitic changes in the röntgen picture.

The *serum phosphatase* values at the first appearance of metaphyseal decalcification in the 12 cases starting in the first 2 weeks of life range between 11.8 and 25.7 units: of the 12 values, 7 are higher and 4 lower than the mean of their age group. In the age group 16—30 days the 17 values from infants given human milk with or without vitamin D range between 12.6 and 61.2 units: the 4 infants of this group who never showed any signs of rickets, had values between 12.6 and 26.7, whereas the remaining 13 values range between 20.4 and 61.2: the mean of these 13 values is 39.8 units, and if this is compared with the mean for infants fed only human milk in the same age group, who had not yet shown any röntgen signs, the difference is 13.0 $\pm$ 4.3 units, i.e. it could have been termed statistically significant had the number of determinations been somewhat higher. — The single infant given cow's milk had a value of 13.5 units, and the few values in higher age groups range between 28.0 and 35.6, i.e. not far from the means of their age groups.

The *serum inorganic phosphorus* in the first two weeks of life range between 4.25 and 6.55 mg %: 5 of the 7 values are higher, and only one lower than the mean of their age group. The 16 values in the age group 16—30 days range between 2.70 and 6.25 mg %: the 4 infants who never showed any signs of rickets, range between 4.20 and 6.10 mg %; for the remaining 12, the mean is 4.23 $\pm$ 0.352 mg % which is nearly the same as the mean for infants given only human milk in this age group who had not yet shown any röntgen changes. — The infant given cow's milk had 5.7 mg %, and in the higher age groups the values ranged between 3.00 and 3.80 mg % for those given human milk with or without vitamin D, and between 4.60 and 5.30 mg % for those given a supply of glucofos.

The *serum calcium* values in the first two weeks range between 6.8 and 13.8 mg % and in the age period 16—30 days

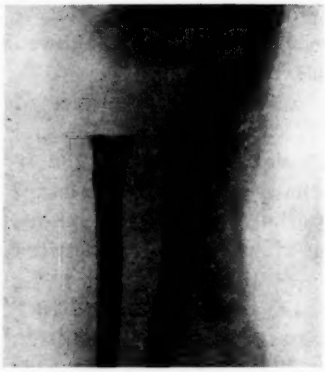


Fig. 4. Born — 24/11 45, birth weight — 1830 gms. Feeding — human milk + D-vimin 10 000 I. U. daily from 25/11 on. — Exam. 28/12 45: Metaphyseal decalcification at the distal ends of ulna and radius.

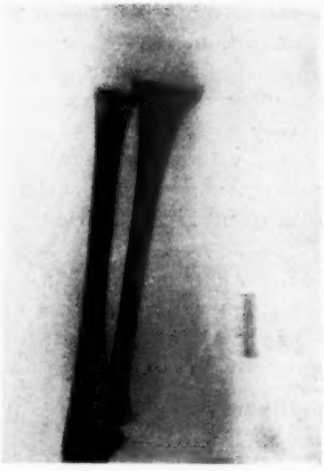
between 6.4 and 12.0 mg %. For the second month of life, the range is 9.1—10.7 mg %. The mean for those given only human milk in the age period 16—30 days is the same as the mean for the infants in that age group which had not yet shown any röntgen changes.

From this it is clear that metaphyseal decalcification is followed in most cases by röntgenological signs of rickets at the subsequent examinations. On the other hand, röntgenological signs of rickets are in most cases preceded by metaphyseal decalcification: in 28 cases out of 40 in the selected series. The serum values at its appearance are about the average of the age group and the feeding group; possibly, the serum phosphatase in the age group 16—30 days is somewhat increased in those infants who will subsequently show röntgen signs of rickets.

Lindblom (1939) was of the opinion that metaphyseal decalcification should not be regarded as a symtom of rickets in the usual sense, as it was not influenced by vitamin D treatment. However, even sure röntgenological signs of rickets may develop under vitamin D treatment.

Obviously, metaphyseal decalcification may very easily escape observation. In a somewhat under-exposed röntgenogram, e.g., it may be impossible to discern it; and its duration

a



b

Fig. 5. — Born 22/5 45, birth weight — 1800 gms. Feeding — human milk, from 11/6 + D-vimin 10 000 U. I. daily, and from 10/7 + citric acid cow's milk 200—250 gms daily.

a) — Exam. 1/6 45: Metaphyseal decalcification at the distal ends of ulna and radius.

b) — Exam. 10/7: Cortical spurs and spreading at radius and ulna, fringing at ulna, and incipient calcification at the epiphyse line of the ulna.

c) — Exam. 23/7: Fringing has disappeared, spreading and spur formation remain; increased calcification at the epiphyse line.

c

is short, so in most cases it was not observed at more than one examination. It is quite possible, therefore, that in reality in all cases of rickets metaphyseal decalcification is present before the other röntgen signs appear. If so, it must be regarded as a very early sign of rickets, even preceding the changes in serum values. Under all circumstances, however, it is not pathognomonic for rickets: some cases which do not show subsequent signs of rickets, are probably not rachitic.

2. *Transverse lines* in the shaft, running parallel to the epiphyse line at some distance from it, have occurred in 7 of the infants selected: they were first observed in the age group 31—45 days, three times, 46—60 days, three times, and 61—75 days, once. The previous feeding had been only human milk in one case; human milk + vitamin D, in one case; cow's milk for 9 days, in one case; and human milk + mineral preparations, in the remaining 4 cases. In three cases the sign was preceded by metaphyseal decalcification; in two of these and in one other, it was preceded by other röntgen changes: spur formation, fringing, calcification at the epiphyse line. In three cases, other signs appeared coincidentally with the transverse lines, but in no case did other signs appear after the transverse lines. In three of the seven cases, no other röntgen changes ever appeared throughout the observation time.

The serum phosphatase values range between 13.3 and 58.8 units. The serum inorganic phosphorus ranges between 4.50 and 6.55 mg %: in 6 cases it was more than 5 mg %. The serum calcium, finally, ranges between 7.4 and 10.4 mg %.

Asada (1924) and also Eliot et al. (1927) interpret the transverse lines as due to a temporary growth retardation, e.g. in children who have had wasting diseases or have been malnourished for long periods of time. Certainly, in the case of premature infants, the possibility of such temporary growth retardations may never be quite denied as they can have been concealed by latent edemas; they may also have occurred prenatally. On the other hand, in these cases no growth retardations were demonstrable; in one case, there had been some

small furuncles of the skin but all the other infants had been healthy except for their debility.

In those cases where the transverse line was preceded by metaphyseal decalcification, it seemed to appear just proximally to the decalcification zone. Possibly it may be interpreted as a healing stage of the metaphyseal decalcification. Anyhow, there seems to be no reason to connect it with rickets.

3. "*F r i n g i n g*" occurred in 17 infants. From Table 36 it can be seen that its first appearance in most cases was in the second month of life, but in a few cases it appeared earlier (in one case at the age of 14 days). In one case it appeared much later, in an infant fed cow's milk for a long time and running in many respects a different course from the other rachitic cases in this material. All the other infants were fed human milk, 6 of them with a supply of vitamin D for between 6 and 45 days (in two cases more than 25 days).

In 11 cases the fringing was preceded by other röntgenological changes, in 8 of them by metaphyseal decalcification, in 3 cases by spur formation, and in one case, the one appearing late after cow's milk feeding, by spur formation, cupping, spreading and calcification at the epiphyse line. In 10 cases other changes appeared at the same examination as fringing (metaphyseal decalcification, once, spur formation, 6 times, spreading, cupping, and calcification at the epiphyse line, each twice). In four cases, however, no other röntgen changes had appeared whether previous to or coincidentally with the fringing. But in these cases, as in 9 others, other changes occurred later (most often calcification at the epiphyse line, and spur formation).

The *serum phosphatase* values in the infants fed human milk with or without vitamin D, range between 19.4 and 68.0 units, with a mean (for the age group 16—75 days) of 41.247 $\pm$ 4.397 units. The difference from the mean for the total group of premature infants fed only human milk may possibly be termed statistically probable as it exceeds twice its standard error. The

Age in days	Total no.	Birth weight			Feeding					Further röntgen changes			
					human milk		cow's milk		mineral suppl.				
		1010—1500	1510—1750	1760—2000	no D	+ D	no D	+ D		preceding	coincident	subsequent	none
7—15	1	1	—	—	—	1	—	—	—	—	—	1	—
16—30	2	—	2	—	1	1	—	—	—	—	—	2	—
31—45	7	2	4	1	5	2	—	—	—	5	5	7	—
46—60	5	1	1	3	4	1	—	—	—	4	4	2	—
61—75	1	—	1	—	—	1	—	—	—	1	—	1	—
106—180	1	—	—	1	—	—	—	1	—	1	1	—	—
7—180	**17**	**4**	**8**	**5**	**10**	**6**	—	**1**	—	**11**	**10**	**13**	—

Table 36. First appearance of "fringing".

infant fed cow's milk had a phosphatase value of 114.4 units, which is the highest value obtained in this material.

The *serum inorganic phosphorus* in the infants fed human milk with or without vitamin D, ranges between 2.05 and 5.50 mg %, with a mean of 3.714 ± 0.323 mg %. The difference from the mean of the total group fed only human milk may possibly be termed statistically probable as it exceeds twice its standard error. In the 11 cases where the fringing had been preceded only by metaphyseal decalcification or by no other changes, the range was 2.05—4.75 mg %. — The infant fed cow's milk had a phosphorus value of 6.15 mg %.

The *serum calcium* in the infants fed human milk without vitamin D, ranges between 7.6 and 11.3 mg %, with a mean of 9.60 mg %. In the infants given vitamin D the range was 9.5—11.1 mg %, and the infant fed cow's milk had a serum calcium of 10.8 mg %.

Thus it seems as if "fringing" is one of the earliest signs of rickets in premature infants, preceded only by metaphyseal decalcification and in some instances by spur formation. The serum phosphatase values at its appearance may possibly be regarded as increased, and the serum inorganic phosphorus values as decreased, in comparison with the general mean for premature infants given only human milk.

This conclusion is in agreement with that of Davidson and Merritt (1934) who regard fraying of the proximal end of the ulna as an important early sign. On the other hand, Eliot and Park (1945) seem to consider it fairly late since they have established that, when judged by microscopic standards, rickets must be at least moderately developed for fraying to be present.

4. *"Cortical spurs"* occurred in 37 of the selected infants. From Table 37 it will be seen that most cases appeared in the age period 31—45 days but in several cases it was observed already at the end of the first month of life. 18 infants had been fed human milk without supplement and 9 human milk + vitamin D for between 6 and 32 days (in 6 cases for 25 days or more). 6 infants had been given cow's milk, usually for 14 days (in one case for 90 days), one of the these also vitamin

Age in days	Total no.	Birth weight			Feeding					Further röntgen signs			
					human milk		cow's milk		mineral suppl.				
		1010—1500	1510—1750	1760—2000	no D	+ D	no D	+ D		preceding	coincident	subsequent	none
16—30	7	—	2	5	5	1	—	—	1	3	3	3	—
31—45	21	3	10	8	9	8	3	—	1	15	11	16	—
46—60	6	—	3	3	3	—	1	1	1	5	5	3	—
61—75	2	1	1	—	1	—	—	—	1	2	1	2	—
106—180	1	—	—	1	—	—	1	—	—	1	—	1	—
16—180	**37**	**4**	**16**	**17**	**18**	**9**	**5**	**1**	**4**	**26**	**20**	**25**	—

Table 37. First appearance of "cortical spurs".

D for 43 days. Three infants had been given glucofos for from 7 to 45 days, and one cow's milk ash for 8 days and vitamin D for 45 days.

In 28 cases the spur formation was preceded by other signs: by metaphyseal decalcification in 24 cases, fringing in 4 cases, and in one case, the one appearing late in an infant fed cow's milk for a long time, by cupping, spreading and calcification at the epiphyse line. At the same examination as the spur formation, metaphyseal decalcification appeared in two cases, fringing in 6 cases, calcification at the epiphyse line in 10 cases, and other signs (spreading, cupping, general decalcification, periosteal encasement of the shaft) in 6 cases. In 4 cases, no other sign had appeared before or coincident with spur formation, and in 11 cases this was so only with metaphyseal decalcification. In 25 cases one or more other röntgen signs appeared at a subsequent examination and in no case was spur formation the only röntgen sign appearing for the whole observation period.

The *serum phosphatase* values range between 13.6 and 83.2 units (in the infants given human milk with or without vitamin D, between 17.5 and 83.2). The mean for the infants given human milk with or without vitamin D, is 39.093 $\pm$ 2.918 units; the difference from the total group of that age fed only human milk can possibly be termed statistically probable as it exceeds twice its standard error. The 5 values in infants given cow's milk or mineral preparations in the same age group range between 26.8 and 40.8, i.e. values rather for infants fed human milk than for cow's milk fed infants. In the higher age groups the values are on an average a little lower.

The *serum inorganic phosphorus* in the infants fed human milk with or without vitamin D ranges between 2.05 and 7.00 mg %, with a mean of 4.143 $\pm$ 0.292 mg %, i.e. not significantly lower than the mean for the total group fed only human milk in that age period. In the infants given cow's milk or mineral preparations, the values range between 5.15 and 6.85 mg %, with a single value as low as 3.65.

Finally, the *serum calcium* values, in the infants given no

vitamin D, range between 6.7 and 11.3, and in those given vitamin D, between 8.8 and 11.6 mg %.

From this it seems as if spur formation is usually one of the earliest röntgen signs of rickets in premature infants, preceded only by metaphyseal decalcification and, in a few cases, by fringing. However, other röntgen signs which generally appear at later stages of rickets, such as calcification at the epiphyse line, seem to coincide with the spur formation more often than with fringing, and this possibly may be connected with the observation that serum inorganic phosphorus is probably not decreased in comparison with the mean for the total series of infants given only human milk. Serum phosphatase, however, is still possibly increased as compared with the total mean. Thus, probably spur formation may be regarded as a somewhat later sign of rickets than fringing. — This opinion seems to agree with that of Eliot and Park (1945).

5 *"Cupping"* occurred in 8 of the infants: in 3 given only human milk, in 3 given human milk + vitamin D for 15—56 days, and in 2 given cow's milk for 8 and 75 days. As will be seen from Table 38, it occurred most often in the second month of life, but later in the cases given cow's milk.

Only in one case was cupping not preceded by any other röntgenological sign: in this case, however, it appeared coincidentally with spreading and calcification at the epiphyse line, so there may have been some changes before which had escaped observation. In the other 7 cases one or more other röntgenological changes had preceded the cupping: metaphyseal decalcification in 5 cases, fringing in 3 cases, spur formation in 5 cases, spreading in 2 cases, and calcification at the epiphyse line, in one case. Coincident with the cupping were fringing in 2 cases, spur formation in one case, spreading in one case, and calcification at the epiphyse line in 4 cases.

The *serum phosphatase* at the first appearance of cupping was, in the infants fed ony human milk, between 44.8 and 57.4 units, in those given human milk + vitamin D, between 14.6 and 29.0, and in those given cow's milk, 18.4 and 44.4

Age in days	Total no.	Birth weight			Feeding					Further röntgen signs			
		1010—1500	1510—1750	1760—2000	human milk		cow's milk		mineral suppl.	preceding	coincident	subsequent	none
					no D	+ D	no D	+ D					
31—45	1	—	1	—	1	—	—	—	—	1	1	1	—
46—60	4	1	1	2	1	3	—	—	—	4	3	2	—
61—75	1	1	—	—	1	—	—	—	—	1	1	1	—
76—105	2	1	—	1	—	—	2	—	—	1	1	1	—
31—105	**8**	**3**	**2**	**3**	**3**	**3**	**2**	—	—	**7**	**6**	**5**	—

Table 38. First appearance of "cupping".

units. The *serum inorganic phosphorus* in the infants given only human milk was 2.90 and 3.70 mg %, in those given human milk + vitamin D, 4.25 and 5.20, and in those given cow's milk, 5.40 and 7.60 mg %. The *serum calcium* ranged between 7.7 (in an infant given cow's milk) and 12.3 mg % (in one given human milk + vitamin D).

As cupping in most cases was coincident with or even subsequent to calcification at the epiphyse line, which is generally accepted as a sign of healing in rickets, and in no case was among the first signs, it must be regarded as a fairly late sign of rickets. The serum values at its first appearance show a very great variation and do not permit any conclusions.

6. "*Spreading*" occurred in 7 of the infants: 2 given only human milk, 3 given human milk + vitamin D for 30—32 days, one given cow's milk for 75 days, and one given glucofos for 25 days. In most cases it appeared in the second month of life.

In three of the cases, the spreading was preceded by metaphyseal decalcification, and in a fourth case this coincided with the spreading. In 4 cases other röntgen signs had preceded the spreading (fringing, spur formation, calcification at the epiphyse line). In all cases some other röntgen sign coincided with the spreading (metaphyseal decalcification, transverse lines, fringing, spur formation, cupping, general decalcification, calcification at the epiphyse line), and in 5 of them other signs appeared at later examinations. Calcification at the epiphyse line once preceded, twice coincided with, and in 4 cases was subsequent to the spreading.

The *serum phosphatase* values range between 28.0 and 58.0 units. The *serum inorganic phosphorus* ranges between 3.60 and 7.00 mg %, and the *serum calcium* between 7.7 and 10.9 mg %.

Spreading, in this series, seems not to be either a very early or a very late sign of rickets. Moreover, it is not "a sufficiently well-defined sign to be valuable by itself" (Eliot and Park, 1945).

Age in days	Total no.	Birth weight			Feeding					Further röntgen signs			
		1010—1500	1510—1750	1760—2000	human milk		cow's milk		mineral suppl.	preceding	coincident	subsequent	none
					no D	+ D	no D	+ D					
31—45	4	1	1	2	1	3	—	—	—	3	4	4	—
46—60	2	1	1	—	1	—	—	—	1	2	2	—	—
76—105	1	—	—	1	—	—	1	—	—	—	1	1	—
31—105	**7**	**2**	**2**	**3**	**2**	**3**	**1**	—	**1**	**5**	**7**	**5**	—

Table 39. First appearance of "spreading".

7. *"General decalcification"* of a pronounced degree was recorded in but the three of the cases selected, in the second and third month of life and always preceded by many other röntgen signs. It was previous to calcification at the epiphyse line only in two cases, in the third case coincident with it. It occurred in one infant given human milk + vitamin D for 15 days, in one given cow's milk for 11 days, and in one given glucofos for 15 days. The serum phosphatase was between 35.8 and 47.2 units, the serum inorganic phosphorus between 2.80 and 5.15 mg %, and the serum calcium between 7.7 and 11.3 mg %. — The picture recorded as general decalcification in these cases certainly represents a much more advanced degree of decreased density than what Lindblom (1940) means by this term and by him is interpreted as an early sign of rickets.

8. *"Periosteal encasements"* of the shafts occurred in 7 infants, in most of them at the end of the second month of life. Two of the cases were given only human milk, two cow's milk for 8 and 15 days + vitamin D for 30 and 43 days, and three cases were given mineral preparations: in two cases cow's milk ash for 5 and 8 days, in one of them + vitamin D for 30 days, and in one case secondary calcium phosphate for 24 days some weeks before and vitamin D for 28 days.

In all the seven cases some other röntgen changes had preceded the periosteal encasements: metaphyseal decalcification in 6 cases, fringing and spur formation each in 4 cases, cupping and spreading each twice, and calcification at the epiphyse line in 3 cases. In 2 cases spur formation, and in 3 cases calcification at the epiphyse line were coincident with the periosteal encasements. In no case did any further röntgen signs appear at subsequent examinations.

The *serum phosphatase* was high in the cases fed only human milk, 68.8 and 53.2 units, and low in those given cow's milk + vitamin D, 13.6 and 16.0 units. In those given mineral preparations it was fairly high, from 33.6 to 40.8 units.

The *serum inorganic phosphorus* in an infant fed only human

Age in days	Total no.	Birth weight			Feeding					Further röntgen signs			
					human milk		cow's milk		mineral suppl.	preceding	coincident	subsequent	none
		1010—1500	1510—1750	1760—2000	no D	+ D	no D	+ D					
31—45	1	—	1	—	—	—	—	—	1	1	1	—	—
46—60	4	2	1	1	2	—	—	1	1	4	2	—	—
61—75	1	—	1	—	—	—	—	—	1	1	—	—	—
76—105	1	—	—	1	—	—	—	1	—	1	1	—	—
31—105	**7**	**2**	**3**	**2**	**2**	—	—	**2**	**3**	**7**	**5**	—	—

Table 40. First appearance of "periosteal encasements" of the shafts.

milk was 3.95 mg % and in the rest ranged between 4.25 and 7.00 mg %.

The *serum calcium* was fairly high in all cases, between 10.1 and 11.4 mg %.

As periosteal encasements were always among the latest signs to appear, and in all but one case were subsequent to or coincident with calcification at the epiphyse line, this sign may be regarded as a sign of healing.

Malmberg (1945), in contrast to this, has found periosteal proliferations of the shafts to be an early sign of rickets, occurring in the majority of premature infants given no vitamin D, at 4—6 weeks of age, at a stage when there are no signs of rickets at the epiphysis. Usually, however, he found it at the tibia which was not examined in my material. This may explain a part of the difference in results. Besides, in my material the main interest was focused on the wrist, so in some cases changes at the shafts may have escaped observation, especially if they were "almost indistinguishable lines" as in some of Malmberg's cases.

9. *Calcification at the epiphyse line* occurred in 34 of the selected infants. As Table 41 shows it appeared most often in the second month of life but it could appear already at the end of the first month, even in infants fed only human milk. It appeared after all sorts of feeding. Vitamin D had been given for from 14 to 75 days, in most cases for more than 25 days. Cow's milk had been given in 9 cases out of 12 for between 5 and 15 days, in the remaining 3 cases for between 29 and 75 days. Mineral preparations (glucofos, 4 times, whey powder, secondary calcium phosphate, and cow's milk ash + vitamin D, each once) in 6 cases out of 8 had been added for only 6—15 days, in two cases, for 26 and 45 days.

In one case, no other röntgen sign ever appeared during the observation period. In 3 more cases, no other signs had been observed previous to the calcification. In 22 cases it was preceded by metaphyseal decalcification, in 13 cases by fringing, in 20 by spur formation, in 4 by spreading, in 3 by cupping,

Age in days	Total no.	Birth weight			Feeding					Further röntgen signs			
		1010—1500	1510—1750	1760—2000	human milk		cow's milk		mineral suppl.	preceding	coincident	subsequent	none
					no D	+ D	no D	+ D					
16—30	3	—	1	2	2	—	—	—	1	2	3	—	—
31—45	7	2	2	3	1	1	3	—	2	5	6	2	1
46—60	15	1	8	6	3	5	2	2	3	15	8	1	—
61—75	3	1	2	—	1	1	—	1	—	3	—	—	—
76—105	6	3	1	2	—	1	3	1	1	5	2	3	—
16—105	**34**	**7**	**14**	**13**	**7**	**8**	**8**	**4**	**7**	**30**	**19**	**6**	**1**

Table 41. First appearance of calcification at the epiphyse line.

and in 3 by general decalcification. Coincident with the calcification spur formation appeared in 10 cases, fringing in 3, cupping in 3, general decalcification in one, transverse lines in 2, and periosteal encasements in 3 cases. Subsequently, other signs appeared in 5 cases: spur formation, spreading, cupping and transverse lines each once, and periosteal encasement 3 times.

The *serum phosphatase* values in the infants fed human milk with or without vitamin D range between 14.6 and 83.2 units, with a mean of 37.293 $\pm$ 4.779 units. This is a little higher than the general mean for premature infants fed only human milk, but the difference is not statistically significant or probable. In the infants given cow's milk, and mineral preparations, the ranges are 16.0—44.4 and 23.2—56.4 respectively; the means are about the same as the general mean for premature infants fed only human milk and definitely higher than for those fed cow's milk, although no closer statistical analysis could be made in the small series.

The *serum inorganic phosphorus* values in the infants fed human milk with or without vitamin D, range between 3.70 and 5.70 mg %. The mean is about the same as the general mean for premature infants fed only human milk. In the infants given cow's milk the range is 4.00—6.85 mg %, and in infants given mineral preparations, 3.65—6.85 mg %. The means are about the same as the general means of these groups.

The *serum calcium* ranges between 7.4 and 11.5 mg %, somewhat higher in those cases given vitamin D.

Thus, calcification at the epiphyse line is always one of the latest signs appearing in this series. The serum phosphatase, however, is not lower at its appearance than in other premature infants, and the serum inorganic phosphorus not higher. It may be a sign of healing but not of health.

Eliot et al. (1927) have called attention to the fact that the calcification zone "is in reality nothing more than the normal zone of provisional calcification of the cartilage which has been suddenly restored, and owes its visibility in the x-ray plate to the fact that the rachitic zone between it and the end of the

shaft, which is almost free from lime salts, casts a feeble shadow and thus allows the provisional zone to show by contrast. — This zone is visible for only a short period of time, because the contrast is soon effaced as the underlying rachitic zone rapidly becomes impregnated with lime salts." — When thus the general decalcification which makes the calcification visible has disappeared, the values will probably be more normal. However, this was not possible to investigate in this material.

S u m m a r y. Röntgen signs of rickets appear in more than half the examinations of premature infants after the first month of life: in the lowest birth-weight group in all examinations, in the middle group, in two-thirds of them and in the highest birth-weight group, in two-fifths of the examinations. Before the age of one month, röntgen signs of rickets appear occasionally.

Metaphyseal decalcification in most cases precedes the appearance of röntgenological signs of rickets: and most cases of metaphyseal decalcification are followed by röntgen signs of rickets. It then may be interpreted, in most cases, as an early rachitic, or pre-rachitic, symtom. It usually appears in the first month of life. In those cases where it appears in the second half of the first month, the serum phosphatase values are possibly a little higher than the average for the age group, but the other serum values are average.

"Fringing" and "cortical spurs" seem to be the earliest signs of rickets in premature infants, except metaphyseal decalcification. They usually appear in the first half of the second month. At the appearance of fringing, serum phosphatase is possibly somewhat higher, and serum inorganic phosphorus somewhat lower than the average for premature infants. At the appearance of spurs, serum phosphatase is possibly higher than the average, but serum inorganic phosphorus seems to be average.

"Cupping", "spreading" and "periosteal encasements" of the shaft, which were not very often recorded, *were often coincident with or even subsequent to calcification at the epiphyse line,* and may then be regarded as *fairly late signs of rickets in this series.*

Calcification at the epiphyse line was always one of the latest signs of rickets to appear in the observation period. The serum values at its appearance are not more normal, however, than the mean for their age group, and it may then be regarded as *a sign of healing but not of health.*

II. Serum values at röntgen signs of rickets

In this chapter the serum values will be studied when distributed according to the results of the simultaneous röntgen examinations. Since röntgen signs of rickets, except metaphyseal decalcification, have only rarely been noted before the age of one month, this analysis is limited to the age group 31—105 days. Those röntgen pictures have been reckoned as normal which reveal no röntgen changes in infants who have never shown any röntgen signs of rickets, except metaphyseal decalcification; normal-looking pictures of infants who have previously shown signs of rickets have been reckoned as "healed" rickets. Those revealing röntgen signs of rickets, including metaphyseal decalcification, who have at a previous examination within the last three weeks shown a normal picture, or only metaphyseal decalcification, have been reckoned as showing a "first sign" of rickets. Other examinations showing signs of rickets, have been regarded as "later" rickets; the exception was made, however, that in infants given cow's milk who had not before shown any sign of rickets, the finding was regarded as a "first sign" of rickets, even if no previous examination had been carried out, if the rickets had clearly developed during the cow's milk feeding. Only those pictures are reckoned as "healed" where all, or nearly all, signs of rickets have disappeared.

1. *Serum phosphatase.* As will be seen from Table 42, the infants showing normal pictures have serum phos-

Röntgen picture	Serum phosphatase (units)							N	M	σ	εM
	—10	11—20	21—30	31—40	41—50	51—60	61—				
Human milk with or without vitamin D											
Normal	1	21	17	7	—	—	—	**46**	*21.259*	± *6.750*	± *1.006*
Rickets:											
First sign	—	2	8	7	7	2	3	**29**	*39.369*	± *15.522*	± *2.933*
Later	—	5	7	13	9	4	3	**41**	*38.607*	± *15.557*	± *2.460*
"Healed"	—	1	5	1	—	1	1	**9**	*32.911*	± *5.423*	
Cow's milk with or without vitamin D											
Normal	—	23	12	1	—	—	—	**36**	*18.081*	± *5.511*	± *0.931*
Rickets:											
First sign	—	2	1	1	2	—	—	**6**	*23.364*	± *10.877*	± *2.093*
Later	2	10	6	2	2	—	—	**22**			
"Healed"	1	5	2	—	—	—	—	**8**	*15.087*	± *3.657*	

Table 42. Serum phosphatase in premature infants 31—105 days old distributed according to the röntgen findings.

phatase values which in almost all cases range between 11 and 30 units, and which never exceed 40 units. This is true both for the infants given human milk and those given cow's milk. Indeed, only 2 values out of 82 exceed 35 units, so higher values than 35 units in a premature infant may certainly be considered as indicating rickets. The difference between the normal values in infants given human milk and in those given cow's milk is not statistically significant, but may possibly be regarded as probable as it exceeds twice its standard error.

Where röntgen signs of r i c k e t s are found in infants given h u m a n milk the serum phosphatase values are generally much higher. Most of them are higher than 30, and almost all of them exceed 20 units. There is no difference between "first sign" of rickets and "later" cases. The few values for "healed" rickets are very varied; this may indicate that the healing is not yet complete, or so recent that the phosphatase values have not yet become normal.

Where rickets is found in infants given c o w's milk, on the other hand, the distribution of the phosphatase values is quite different. Here, most values are lower than 30 units, and some of them are extremely low. The few cases showing "first signs" of rickets, however, have generally somewhat higher values, with a mean of 29.233 units. The mean for the rickets values is a little higher than for the normal values in cow's milk-fed infants; the difference is not statistically significant but can possibly be termed probable as it exceeds twice its standard error. — The few values for "healed" rickets here are very low.

2. *S e r u m i n o r g a n i c p h o s p h o r u s.* It appears from Table 43 that in infants giving n o r m a l röntgen pictures the phosphorus values in almost all cases exceed 4 mg %; values below 4 mg % may thus be regarded as indicating rickets. The mean for normal values in infants fed human milk is lower than for normal values in infants fed cow's milk; the difference exceeds 2 1/2 times its standard error and may be regarded as statistically probable.

When signs of r i c k e t s are present in infants fed h u m a n

Röntgen picture	Serum inorganic phosphorus (mg%)						N	M	σ	εM
	2.05—3.00	3.05—4.00	4.05—5.00	5.05—6.00	6.05—7.00	7.05—				
Human milk **with or without vitamin D**										
Normal	—	3	10	20	6	3	**42**	*5.438*	± *0.989*	± *0.155*
Rickets:										
First sign	3	10	6	4	—	—	**23**	*3.933*	± *0.849*	± *0.181*
Later	3	7	10	7	1	—	**28**	*4.396*	± *1.081*	± *0.165*
"Healed"	—	2	—	3	—	—	**5**	*4.730*		
Cow's milk										
Normal	—	—	4	14	10	7	**35**	*6.090*	± *0.947*	± *0.163*
Rickets:										
First sign	—	—	—	3	3	—	**6**	*5.850*	± *0.957*	± *0.209*
Later	—	1	3	7	3	2	**16**			
"Healed"	—	—	—	2	3	2	**7**	*6.686*		

Table 43. Serum inorganic phosphorus in premature infants 31—105 days old distributed according to the röntgen findings.

milk, the values are generally much lower, and this is especially the case at the "first signs" of rickets; the difference between "first sign" and "later" rickets is, however, not significant. In most cases of rickets, the values are lower than 5 mg %, and in nearly half the series they are lower than 4 mg %. There are, however, exceptions, and values may be seen as high as 5.70 mg % and, in one single case, 7.00 mg %. — The few values in "healed" rickets vary; in some instances they are normal, in some others, fairly low.

The rickets values in infants given cow's milk, on the other hand, are quite differently distributed. Here, almost all values are higher than 5 mg % and even extremely high values may be obtained. The mean is not significantly lower than the "normal" in premature infants given cow's milk. Thus, in premature infants given cow's milk, the serum inorganic phosphorus value cannot be used as a symtom of rickets in the first months of life. — The values are high in the few cases of "healed" rickets in cow's milk-fed infants.

3. *Serum calcium.* Here the values have been divided into four groups, one given vitamin D and one given no vitamin D in each feeding group. From Table 44 it may be seen that when the röntgen findings are normal, most cases range between 9 and 11 mg %. The means for three of the groups are almost the same, but for the group given cow's milk but no vitamin D, it can possibly be regarded as lower than in the other groups, as the differences from them are more than twice their standard errors, and from the group given cow's milk + vitamin D more than 2 1/2 times the standard error.

When signs of rickets are present in infants given no vitamin D, the values are lower than with normal röntgen findings. The differences are not statistically significant, but for the human milk groups may be regarded as statistically probable as it exceeds 2 1/2 times the standard error. Most values range between 8.0 and 10.0 but in some cases are as low as 6.4 mg %.

When vitamin D was given, on the other hand, the

Röntgen picture	Serum calcium (mg%)							N	M	σ	εM
	6.1—7.0	7.1—8.0	8.1—9.0	9.1—10.0	10.1—11.0	11.1—12.0	12.1—13.0				
Human milk no vitamin D											
Normal	—	—	1	9	8	2	—	**20**	*10.240*	± *0.650*	± *0.149*
Rickets:											
First sign	1	1	1	9	2	3	—	**17**	*9.500*	± *1.225*	± *0.220*
Later	1	1	2	7	4	—	—	**15**			
"Healed"	—	—	—	—	2	—	—	**2**	*10.500*		
Human milk + vitamin D											
Normal	—	—	2	6	16	6	—	**30**	*10.333*	± *0.817*	± *0.152*
Rickets:											
First sign	—	—	—	2	4	3	—	**9**	*10.650*	± *0.758*	± *0.128*
Later	—	—	—	7	14	4	2	**27**			
"Healed"	—	—	—	—	3	2	—	**5**	*10.820*		
Cow's milk no vitamin D											
Normal	—	2	3	8	4	3	—	**20**	*9.675*	± *1.039*	± *0.238*
Rickets:											
First sign	—	1	1	—	1	—	—	**3**	*8.889*	± *1.348*	± *0.477*
Later	—	2	1	2	—	1	—	**6**			
"Healed"	—	—	—	2	2	—	—	**4**	*10.250*		
Cow's milk + vitamin D											
Normal	—	—	1	4	11	1	1	**18**	*10.522*	± *0.895*	± *0.217*
Rickets:											
First sign	—	—	—	—	2	1	—	**3**	*10.561*	± *0.773*	± *0.165*
Later	—	—	2	2	10	6	—	**20**			
"Healed"	—	—	—	1	3	—	—	**4**	*10.225*		

Table 44. Serum calcium in premature infants 31—105 days old distributed according to the röntgen findings.

Table 44. Serum calcium in premature infants 31—105 days old distributed according to the röntgen findings.

serum calcium values are the same when there are röntgen signs of rickets as when the röntgen findings are normal.

4. *Serum protein.* When the röntgen findings are normal in infants fed human milk, the serum protein is 5.065 ± 0.091 %, as against 4.967 ± 0.075 % when signs of rickets are present; the difference is not significant. In infants given cow's milk, it is 5.333 ± 0.079 % when the röntgen findings are normal, and 5.006 ± 0.117 % when rickets is present; here the difference can possibly be regarded as statistically probable as it exceeds twice its standard error.

Thus, *when röntgen findings are normal,* the serum values in most cases in premature infants differ only moderately in the age period 31—105 days from the values obtained in the normal material. The serum phosphatase in the infants fed human milk is somewhat higher, and this difference is statistically significant but it is less than 5 phosphatase units. In the infants given cow's milk, the phosphatase values are almost the same as normal.

The serum inorganic phosphorus in the infants given cow's milk is also about normal, but the standard deviation is greater than normal. In infants fed human milk, the values have decreased significantly, to about 0.8 mg % below normal; here, too, the standard deviation is greater than normal.

The serum calcium, on the other hand, is significantly lower than normal in all groups, and this is especially the case in the group given cow's milk but no vitamin D.

When röntgen signs of rickets are present, the serum phosphatase and serum inorganic phosphorus values are extremely abnormal in the premature infants given human milk. The serum phosphatase here is more than twice as high as normal; and the standard deviation is more than three times the normal. The serum inorganic phosphorus is less than two-thirds of the normal, and its standard deviation is nearly twice the normal.

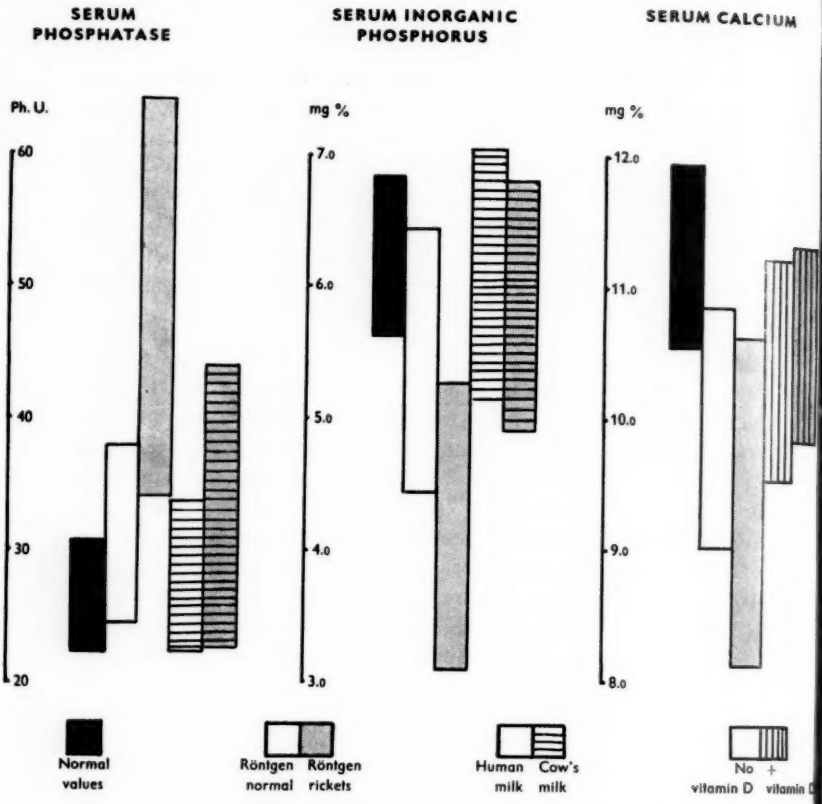


Fig. 6. Premature infants, birth weight 1010—2000; serum values at 31—105 days of age when röntgen findings are normal and when signs of rickets are present, with different feedings.

(Note that in serum phosphatase and serum inorganic phosphorus the blank columns represent human milk feeding with or without vitamin D, but in serum calcium means there has been no vitamin D supply with human or cow's milk).

In the infants given cow's milk, on the other hand, the phosphatase and phosphorus values are much more normal. The serum phosphatase is higher, and even higher than in premature infants fed cow's milk who show normal röntgen pictures; the difference from them can possibly be regarded as statistically probable, as it is more than twice its standard error. The standard deviation is also higher: more than twice the normal. The serum inorganic phosphorus, however, is not significantly less than normal. Its standard deviation is still somewhat higher.

The serum calcium, finally, is the same as when röntgen findings are normal in the infants given human milk or cow's milk plus vitamin D, but definitely lower in the groups given no vitamin D. For the group given cow's milk but no vitamin D, the difference is not significant, but this may depend only upon the small number of examinations in that group.

Neither a supply of vitamin D nor of cow's milk is certain to prevent rickets. It must be admitted, however, that most cases of rickets in the cow's milk group had appeared before any cow's milk had been given; only in 6 infants had cow's milk been given before the first sign of rickets appeared, and in two of these cow's milk had been given from the second week of life and vitamin D 10 000 I.U. daily from the first day of life. *Even where rickets is not prevented, there is generally an effect of the supply on the serum values; i.e., vitamin D has increased the serum calcium, and cow's milk has increased the serum inorganic phosphorus and decreased the serum phosphatase even in the cases where rickets has developed.*

Summary. — *The serum phosphatase is much higher and the serum inorganic phosphorus much lower, when röntgen signs of rickets are present than when röntgen findings are normal in infants fed human milk. In infants fed cow's milk, no significant difference in these values can be obtained between*

rickets and normal findings; the values are fairly good in both cases.

The serum calcium is much lower when röntgen signs of rickets are present than when röntgen findings are normal in infants given no vitamin D. If vitamin D is given, the serum calcium is about the same whether or not rickets is present.

Cow's milk improves the serum phosphatase and the serum inorganic phosphorus, even where it cannot prevent röntgen signs of rickets; and vitamin D improves the serum calcium even in the cases where rickets has developed.

Discussion

The following facts have been established by this investigation:

Premature infants with a birth-weight not above 2 000 gms and *fed exclusively human milk* in the first months of life will develop an increased serum phosphatase, decreased serum inorganic phosphorus and serum calcium and, very frequently, röntgen signs of rickets. When röntgen signs of rickets are present, all the serum values are more abnormal than when they are absent.

A *supply of vitamin D* will increase the serum calcium; in the highest birth-weight group, 1 760—2 000 gms, it may also improve the serum phosphatase and serum inorganic phosphorus a little. It does not prevent the development of röntgen signs of rickets. In the presence of such signs, the serum phosphatase and serum inorganic phosphorus are more abnormal than in their absence, but serum calcium now is as high as when röntgen is normal.

A *supply of cow's milk* will decrease the serum phosphatase and increase the serum inorganic phosphorus to about normal values; on the other hand, it does not improve the serum calcium. Röntgen signs of rickets seem to occur rather rarely when cow's milk is given; and then serum phosphatase is as low and serum inorganic phosphorus as high as when röntgen is normal, but serum calcium seems to be still more decreased, provided no vitamin D is given at the same time. — The same effect as of cow's milk seems to be obtained, at least in some degree, by giving *mineral preparations* e.g. calcium

hexose-diphosphate, so the effect seems to be caused by the mineral content of the cow's milk.

From this it may be concluded that:

The phosphorus requirement of the premature infant is not satisfied when only human milk is given;
and the calcium requirement only if vitamin D is supplied:

and even then, the calcium requirement seems to be less completely satisfied than in full term infants, as the serum calcium never reaches normal values.

A supply of cow's milk may fulfil the phosphorus requirement,
but not the calcium requirement if no vitamin D is supplied.

The bone phosphatases, which are supposed to hydrolyze the hexose-phosphoric esters of the body fluids (Robison et al. 1924) *may be assumed to increase their activity as a response to the increased difficulty of fulfilling their function,* and this is reflected in the increased serum phosphatase found when there is a deficit in phosphorus in the body. This may indicate that in premature infants given only human milk, not only the serum inorganic phosphorus is decreased but also the organic phosphorus compounds upon which the phosphatase acts; this was known before concerning rickets (Guest and Rapoport 1941).

Rickets seems to occur most often when the phosphorus supply is most scanty,
but it may occur even when there is an ample supply of phosphorus.
Rickets is possibly also most frequent when the calcium supply is scanty,
but it is frequent even when there is an ample supply of calcium.

The possible causes of the lack of calcium and phosphorus may be:

An insufficient storage at birth;
An insufficient supply with the food;
An insufficient absorption from the intestine, or increased excretion;
Increased demands, e.g. by rapid growth.

An insufficient storage at birth of calcium and phosphorus is characteristic of premature infants, according to the investigations of Givens and Macy (1933) and of Iob and Swanson (1934; cf. p. 11). It is due mainly to the fact that most of the calcium and phosphorus is stored in the body during the last months of intra-uterine life. It can be imagined, however, that the mineral content of the foetus may also depend, to some extent, on the maternal care during pregnancy. This question, of course, is very difficult to investigate. In my material, no significant difference could be proved between the occurrence of rickets in infants whose mothers were reported to have taken vitamin D for at least a month during pregnancy, and those whose mothers were reported not to have taken any vitamin D. This, of course, will not permit any conclusions.

An insufficient supply with the food is suggested, for c a l c i u m, e.g. by Leitch (1937; cf. p. 11). To satisfy the demands proposed by him, it would be necessary to give the infants an average amount of human milk of about 430 ml. daily during the first month of life, and of more than 1 litre daily during the second month of life even assuming that all the calcium would be retained. This of course is impossible. Even if the demands are reduced to half those proposed by Leitch with regard to the slower absolute increase in weight of the premature infants, it would not be possible to satisfy them with human milk, because all the calcium supplied is not retained in the body.

An insufficient supply of p h o s p h o r u s was proved by Benjamin et al. (1943; cf. p. 12) in premature infants given human milk.

In my material it was not possible to demonstrate any difference between the serum values in infants who had consumed large quantities of human milk per gm of weight increase, and

in those who had consumed smaller quantities, in the age period 31—105 days.

An insufficient absorption from the intestine in premature infants fed human milk was proved, for calcium, by Benjamin et al. (1943) who stated that this must depend on the insufficient supply of phosphorus; the phosphorus, on the other hand, was almost completely absorbed. Nicolaysen (1937) has established the fact that in rats the absorption of calcium is greatly dependent upon the presence of vitamin D; the phosphorus absorption is not dependent, directly, on vitamin D, but secondarily it is delayed to a certain extent in the absence of vitamin D, because the phosphorus then will precipitate in the intestine as a consequence of the increased amount of non-absorbed calcium present there.

The relatively rapid growth in premature infants is listed by many among the factors which cause the increased occurrence of rickets in them. "Certainly the reverse is true, that an arrest in growth tends, though not invariably, to heal or prevent the development of the disease (Barnes 1945). The same is the case with the *increased incidence of infection and digestive disturbances* (e.g. Crosse 1945; cf. p. 14).

In my material it was not possible to prove any difference between the serum values in rapidly growing infants and in those growing more slowly of each feeding group in the age period 31—105 days. — It might be suspected that the abnormal serum values obtained in the infants given only human milk, depend in some degree on the fact that the infants who fall ill or who die in the hospital, generally belong to this group. However, when all infants are excluded who have shown any signs of ill-health except debility or who have died in the hospital, the means for all the serum values are still the same. On the other hand it is remarkable that all infants who become ill or who die after the first month of life almost without exception, lack röntgen signs of rickets. This may possibly be explained by the growth retardation due to the illness.

It seems as if my results may be explained fairly well in the light of the investigations just quoted by Benjamin et al. (1943) and by Nicolaysen (1937):

The premature infant receives in human milk an insufficient amount of phosphorus for the demands of its skeleton, and a fairly sufficient amount of calcium, but the calcium will not be absorbed in a sufficient quantity unless vitamin D is supplied. When cow's milk is supplied, a sufficient amount of phosphorus will be absorbed, but not a sufficient amount of calcium, in spite of the large supply in the intestine, unless vitamin D is present.

Rickets in the first months of life in premature infants is most often due to an insufficient supply of phosphorus with the food, or an insufficient absorption of calcium. But it may occur, occasionally, even when both minerals seem to be absorbed in sufficient amounts, so this may not be the only cause of early rickets in premature infants.

Finally, it remains to be proved that the röntgen signs of rickets obtained at this investigation, are really signs of the same anatomical changes as are usually defined as rickets. This, of course, requires anatomical investigations. These are planned, and will be carried out as a subsequent part of this work.

Summary

All premature infants with a birth-weight not above 2 000 gms born at the Sahlgren's Hospital Obstetric Department during 2 1/2 years and whose debility was not too grave to permit examination, were examined at regular intervals with blood analyses and röntgen of the wrist. An account of the material is given in Table 1, p. 20.

To get an idea of the *normal values* of the chemical methods used, two normal groups of full term breast-fed infants were examined, one of new-born infants in the obstetric department, the other of infants in the first months of life at a children's welfare centre. The normal values obtained are summarized on p. 38.

Premature infants fed only human milk without any supplement in the first days of life have about the same serum inorganic phosphorus values as normal, higher serum phosphatase, and lower serum calcium and serum protein values. In the following months, the serum phosphatase increases, and the serum inorganic phosphorus and serum calcium decrease considerably. This is summarized on p. 48.

Premature infants fed human milk + vitamin D show significantly higher serum calcium than those given no vitamin D; the infants of the highest birth-weight group investigated, 1 760—2 000 gms, possibly also show somewhat lower serum phosphatase and higher serum inorganic phosphorus than the corresponding group given no vitamin D. This is summarized on p. 56.

Premature infants given cow's milk but no vitamin D show significantly lower values for serum phosphatase, and higher

for serum inorganic phosphorus than those given human milk but no vitamin D. This is summarized on p. 60.

Premature infants given cow's milk + vitamin D show significantly lower values for serum phosphatase and higher for serum inorganic phosphorus and serum calcium than those given human milk without any supplement. The serum phosphatase and serum inorganic phosphorus values are quite normal. This is summarized on p. 66.

Supplies of different mineral preparations may act to a certain extent in the same manner as cow's milk. This is summarized on p. 71.

A comparison of the serum values in the different groups is made on p. 72 and shown graphically in figs. 1—3, p. 73—75.

Seasonal variations in the serum values have been established for the normal groups and for premature infants fed human milk with or without vitamin D. They are summarized on pp. 48, 56 and 60.

Röntgen signs of rickets appear in more than half the examinations after the first month of life; before the age of one month, they appear occasionally. Metaphyseal decalcification, which may possibly be regarded as an early rachitic, or pre-rachitic sign, usually appears before the age of one month. The röntgen findings are summarized on p. 99.

When röntgen signs of rickets are present in premature infants fed human milk with or without vitamin D, the serum phosphatase is much higher and the serum inorganic phosphorus lower than when the röntgen picture is normal. In premature infants fed cow's milk there is no difference in these respects. In premature infants given no vitamin D, the serum calcium is much lower when röntgen signs of rickets are present, but when vitamin D is given, serum calcium is the same in infants showing rickets and in those with normal röntgen findings. This is summarized on p. 109.

It is concluded that human milk feeding may not supply the premature infant with a sufficient amount of phosphorus, though it gives it a fairly sufficient amount of calcium; the calcium will, however, not be absorbed in sufficient amounts

unless vitamin D is given. When cow's milk is supplied, a sufficient amount of phosphorus will be absorbed, but not of calcium unless vitamin D is given. Rickets in the first months of life in premature infants is most frequently due to an insufficient supply of phosphorus or an insufficient absorption of calcium, but this may not be the only cause.

References

Asada, T. Über die Entstehung und pathologische Bedeutung der im Röntgenbild des Röhrenknochens am Diaphysenende zum Vorschein kommenden „parallellen Querlinienbildung". — Zbl. f. Kind. 18: 705: 1925.

Bakwin, H. Pathogenesis of tetany of the new-born. — Am. J. Dis. Child. 54: 1211: 1937.

Barnes, D. J. Rickets. — In Litchfield and Dembo. Therapeutics of Infancy and Childhood. Philad. 1945.

Barnes, D. J., and *Munks, B.* Serum phosphatase, calcium and phosphorus values in infancy. — Proc. Soc. exp. Biol. & Med. 44: 327: 1940.

Benjamin, H. R., Gordon, H. H. and *Marples, E.* Calcium and phosphorus requirements of premature infants. — Am. J. Dis. Child. 65: 412: 1943.

Beumer, H. Rachitis und Tetanie. — In Handbuch der Kinderheilkunde v. Pfaundler. Ergänzungsbd I. Berlin 1942.

Buch, I. and *Buch, H.* An improved King and Armstrong method for the determination of phosphatase activity in blood serum. — Acta med. scand. 101: 211: 1939.

Bullock, J. K. The physiologic variations in the inorganic blood phosphorus content at the different age periods. — Am. J. Dis. Ch. 40: 725: 1930.

Crosse, V. M. The premature baby. — London 1945.

Dahlberg, G. Statistical Methods for medical and biological students. — London 1940.

Davidson, L. T. and *Merritt, K. K.* Viosterol in the prophylaxis of rickets in premature infants. — Am. J. Dis. Child. 48: 281: 1934.

Denzer, B. S., Reiner, M., and *Weiner, S. B.* Serum calcium in the new-born. — Am. J. Dis. Child. 57: 809: 1939.

Du Toit, P. J. and *Malan, A. L.* Phosphorus and calcium deficiency diseases as 2 aetiologically distinct entities. — Nature 140: 153: 1937.

Eliot, M. M. and *Park, E. A.* Rickets. — In Brennemann's Practice of Pediatrics, vol. I. 1945.

Eliot, M. M., Souther, S. P. and *Park, E. A.* Transverse lines in x-ray plates of the long bones of children. — Bull. Johns Hopkins Hosp. 41: 364: 1927.

Fiske, C. H. and *Subbarow, Y.* The colorimetric determination of phosphorus. — J. Biol. Chem. 66: 375: 1925.

Gerstenberger, H. J. Rickets in monkeys. — Acta paed. 22: 422: 1938. — Phosphate solutions in the treatment of infantile rickets. — Am. J. Dis. Child. 58: 668: 1939.

Gerstenberger, H. J. and *Nourse, J. D.* The prevention of rickets in premature infants. — J. Am. Med. Ass. 87: 1108: 1926.

Givens, M. H. and *Macy, I. G.* The chemical composition of the human fetus. — J. Biol. Chem. 102: 7: 1933.

Guest, G. M. and *Rapoport, S.* Organic acid- soluble phosphorus compounds of the blood. — Physiol. Rev. 21: 410: 1941.

Hamilton, B. The calcium and phosphorus metabolism of prematurely born infants. — Acta paed. 2: 1: 1922.

Hamilton, B. and *Schwartz, Ch.* Role of acidosis and alkalosis in the etiology of rickets. — Am. J. Dis. Ch. 46: 669: 1933.

Hess, A. F. Rickets including Osteomalacia and Tetany. — London 1929.

— The forms of the calcium and inorganic phosphorus in human and animal sera. — J. Biol. Chem. 100: 27: 1933.

Hickmans, E. M., Finch, E. and *Tonks, E.* Plasma protein values in infants. — Brit. med. Bull. 2: 189: 1944.

Holt, L. E. and *McIntosh, R.* Diseases of Infancy and Childhood. — New York 1939.

Howland, J. and *Kramer, B.* A study of the calcium and inorganic phosphorus of the serum in relation to rickets and tetany. — Mon. Kind. 25: 279: 1923.

Iob, V. and *Swanson, W. W.* Mineral growth of the human fetus. — Am. J. Dis. Child. 47: 302: 1934.

Johnsson, T. Massive dose prophylaxis with vitamin D for prematurely born infants. — Acta paed. 32: 473: 1945.

Jorpes, E. and *Magnusson, H.* An application of Pregl's micro-analysis to the determination of free phosphates in the blood. — Acta paed. 7: 1: 1928.

Josefsson, E. On the values for plasma phosphatase and blood phosphorus in healthy children born at term and in premature children. — Ann. paed. 157: 193: 1941.

Klackenberg, G. Three years' prophylaxis against rickets in prematures, with D_2 massive dose. — Acta paed. 32: 508: 1945.

Lehmann, J. Bestämning av totalprotein i serum enligt Barbour och Hamilton (modifierad av förf.). — NM 21: 321: 1944.

Leitch, I. The determination of the calcium requirements of man. — Nutr. Abstr. & Rev. 6: 553: 1937.

Linblom, K. Early röntgen signs in rickets. — Acta paed. 25: 170: 1939.

Lundsteen, E. and *Vermehren, E.* Micro-methods for the estimation of phosphatases in blood plasma and inorganic phosphorus in blood. — Compt. rend. des trav. du Lab. Carlsberg, sér. chim. 21: 147: 1936.

Mai, H. Körpereigene Ultraviolettstrahlung und Rachitis. Zbl. f. Kind. 32: 303: 1936.

Malmberg, N. Erfahrungen bei der Rachitisprophylaxe frühgeborener Kinder II. — Acta paed. 29: 367: 1942.

— Citridomjölksuppfödning och rachitis. Nord. Med. 16: 3474: 1942.

— Occurrence and significance of early periosteal proliferation in the diaphyses of premature infants. — Acta paed. 32: 626: 1945.

Marfan, A.-B. Nouvelles études sur le rachitisme III. — Le Nourrisson 29: 1: 1941.

Martland, M. and *Robison, R.* Note on the estimation of phosphorus in blood. — Bioch. J. 18: 765: 1924.

Nicolaysen, R. Studies upon the mode of action of vitamin D II—V. —Bioch. J. 31: 107, 122, 323, 1 088: 1937.

Peters, J. P. and *van Slyke, D. D.* Quantitative and Clinical Chemistry. — Baltimore 1935.

Robison, R. and *Soames, K. M.* The possible significance of hexosephosphoric esters in ossification II. The phosphoric esterase in ossifying cartilage. — Bioch. J. 18: 740: 1924.

Robison, R. and *Kay, H. D.* — III. The action of the bone enzyme on the organic phosphorus compounds. — Bioch. J. 18: 755: 1924.

Rominger, E. Über Versuche zur Rachitisheilung und -Verhütung ohne Anwendung von Licht oder D-Vitamin. — Arch. f. Kind. 131: 53: 1944.

Rominger, E. and *Meyer, H.* Die Salzretention des gesunden Brust- und Flaschenkindes. — Arch. f. Kind. 80: 195: 1927.

Shohl, A. T. The effect of low calcium-high phosphorus diets at various levels and ratios upon the production of rickets and tetany. — J. Nutr. 11: 275: 1936.

Shohl, A. T. and *Butler, A. M.* Citrates in the treatment of infantile rickets. — New Engl. J. Med. 220: 515: 1939.

Smith, Cl. A. The physiology of the new-born infant. — Springfield 1945.

Sobel, A. E. and *Kaye, I. A.* Estimation of submicroquantities of calcium. — Industr. & Engin. Chem., Analyt. Ed. 12: 118: 1940.

Stearns, G. Mineral metabolism of normal infants. — Physiol. Rev. 19: 415: 1939.

Stearns, G. and *Warweg, E.* The partition of phosphorus in whole blood and serum, the serum calcium and serum phosphatase from birth to maturity. — J. Biol. Chem. 102: 749: 1933.

Swanson, W. W. The composition of growth. II. The full term infant. — Am. J. Dis. Child. 43: 10: 1932.

Todd, W. R., Chuinard, E. G. and *Wood, M. T.* Blood calcium and blood phosphorus in the new-born. — Am. J. Dis. Child. 57: 1278: 1939.

Tow, A. The premature infant. — Advances in Pediatrics I: 189: 1942.

Vermehren, E. Om Plasmafosfatase hos normale Börn og Voksne samt ved Rachitis og Tetani. — Köbenhavn 1938.

Ylppö, A. Das Wachstum der Frühgeborenen von der Geburt bis zum Schulalter. — Ztschr. f. Kind. 24: 111: 1919.

— Das Skelett der Frühgeborenen. — In Handbuch der Kinderheilkunde v. Pfaundler u. Schlossmann. 4. Aufl. I. Berlin 1931.

From The University Pediatric Clinic
of Crown Princess Lovisa's Hospital for Children, Stockholm
(Head: Professor A. Lichtenstein, M. D.)

GASTRIC JUICE EXAMINATIONS IN CHILDREN

A Clinical-Physiological Study

By

KURT KAIJSER

ACTA PÆDIATRICA, VOL. XXXIII, SUPPLEMENTUM III

Stockholm 1946

UPPSALA 1946
ALMQVIST & WIKSELLS BOKTRYCKERI AB

INTRODUCTION.

This investigation deals with the study of certain physiological and clinical conditions concerning the secretion and composition of gastric juice in children.

It has been carried out mainly at the University Pediatric Clinic in Crown Princess Lovisa's Hospital for Children in Stockholm, Sweden (Head: Professor A. LICHTENSTEIN). Part of the physiological experiments has been made in the Physiological Institute in Uppsala, Sweden (Head: Professor T. TEORELL).

The investigation, which is partly a comprehension of results previously published (KAIJSER, 1943), has been put up in three parts comprising:

PART I. — *Physiological studies of the secretion and composition of gastric juice.*

Determination of the concentration of the acidity of freshly secreted gastric juice from the gastric glands (= primary acidity).

Examination of the acidity-lowering factors in the primary acidity, through which secondary acidity arises, which is found in a gastric juice sample taken from the stomach.

Investigation of the diffusion conditions in the stomach for hydrochloric acid and nitrate of ammonia.

In order to attain the greatest possible experimental precision, the above investigations have been carried out on kittens as well as on children.

PART II. — *Clinical studies of the secretion and composition of gastric juice in healthy children.*

Examination of gastric juice in about 40 healthy children from 5 months to 14 years of age. Special measures have

been observed on these occasions of which the following can be mentioned.

Aspiration of gastric juice has been carried out by a special method which enabled one to obtain an especially pure juice.

Histamine and insulin have been used as a parenteral stimulus in order to obtain as plentiful a flow of gastric juice as possible without admixture with food.

The quantity of gastric juice secreted, free HCl, total acidity and the chloride and pepsin values have been determined.

EWALD'S test meal has been given to compare the results obtained by the procedure mentioned above.

PART III. — *The diagnostic significance of gastric juice examination in children.*

Examination of gastric juice in children suffering from certain stomach and intestinal disturbances.

The importance of variation in the composition of the test meal and the time for its administration.

Suggestion for routine clinical examinations of gastric juice in children.

PART I.

Physiological Studies of Gastric Juice Secretion and Composition.

Primary acidity.

Previous investigations.

Adult individuals. That hydrochloric acid, when it is secreted by the gastric glands, has, in adults, a high acidity of a fairly constant maximum value, primary acidity, is probable. Teorell (1933, 1940) has obtained a primary acidity level of 208 ± 6 mN in grown-up cats[1]. In man this level is thought to lie round about 175 mN HCl (see Elliot, Risholm, Öbrink 1942).

Young individuals. The acidity value in the ventricle of children has been the subject for many investigations. Since the test meal procedure on children was introduced in 1888 it has been relatively simple to obtain gastric juice samples from children of all ages.

The procedure has varied in that one has examined the contents of the ventricle on a fasting stomach as well as after different test meals. The above investigations apply to the secondary acidity but, on the basis of these, one has drawn conclusions concerning the primary acidity as well.

It has been said that an infant is incapable of producing gastric juice with a high hydrochloric acid value. This increases first with years. Some authors are of the opinion that the gastric juice of a child reaches the value of that of an

[1] N = normal = gram equivalent per litre; mN = milli-normal = milli-equivalents per litre.

adult earliest at the age of four years whilst others draw the line of demarcation at 10 years.

My personal investigations.

It was my intention to investigate to what degree the primary acidity of the gastric juice in young individuals, irrespective of their age, is as high as that of adults.

The investigations were carried out on children and kittens.

Procedure.

I have followed the procedure given by TEORELL (1940) which is as follows:

A measured quantity of a buffer solution having a large acid-binding capacity and a scanty permeability through the ventricle mucosa is passed down into the ventricle. TEORELL used a 0.25-M aqueous solution of glycocoll having a pH of about 6.0. Owing to the good buffering qualities of this solution the acid-lowering factors in the ventricle could not, to any great extent, make themselves manifest on the HCl content of the gastric juice (primary acidity) secreted directly into the buffer solution.

By means of parenteral histamine stimulation a flow of gastric juice is obtained and the pH in the solution falls. After about 15 minutes the buffer solution is aspirated in its entirety and the increase in volume measured. The solution is titrated back to pH 6.0 with 0.1-N NaOH after which one can calculate the primary acidity of the hydrochloric acid introduced.

In order to test the accuracy of the measurements, several trial experiments were made as follows:

To 5 ml 0.25-M glycocoll, 1.5 ml 170 mN HCl was added. When titrating back to pH 6.0 the consumption was 2.56 ml 0.1-N NaOH. The calculated value of HCl was therefore $\frac{2.56 \cdot 100}{1.5} = 170.7$ mN (the corrected value was 170 mN).

The procedure therefore gave very reliable values.

pH-measurements were carried out electrometrically with antimony electrodes. The chloride titrations were also done electrometrically.

Primary acidity in children.

The postulate for acceptable results from the above procedure is that the buffer solution, which is introduced into the ventricle, is aspirated as far as possible in its entirety. Examinations were made on children with pylorostenosis in whom the reduced passage through the pylorus could be established by roentgen control.

It was possible to carry out aspiration with an accuracy of about $\pm$ 0.1 ml. The examinations were carried out under a chloral anaesthetic to prevent the children from becoming too restless. They were examined on a fasting stomach.

Several examinations were made but only a few typical examples of the results obtained will be given here.

Case 1. Age 6 weeks. Weight 2960 g. Has had spouting vomiting attacks since the age of four weeks. *X-ray examination:* the contents of the ventricle emptied quite slowly into the intestine and after two hours an insignificant amount of contrast was still found in the small intestine and a large remainder in the ventricle. *Pylorostenosis* (SAHLSTEDT).

7.30 a. m. a retention of 5 ml (free HCl 1, total acidity 44) was aspirated through a rubber tube which had been allowed to remain during the whole of the examination. The ventricle was then washed out several times with 0.25-M glycocoll and aspirated empty. The following experiment was made to calculate the water resorption from the glycocol solution and the stipulated decrease in volume during a 15-minute period.

At 8.10 a. m. 10 ml glycocoll (pH 6.0) was passed down and the whole amount aspirated at 8.25 o'c. It transpired that an admixture of HCl had taken place but, despite this, the volume had not increased. Examination of the aspirated solution and back titration to pH 6.0 gave the following result (tab. 1).

If one counts on a theoretical value of 175 mN for the primary acidity, the total volume should have increased by 0.14 ml. As there was no measurable increase in volume it is prob-

Table 1.

Time	Amount ml	Increase ml	pH	ml 0.1-N NaOH	H mN	ml 0.1-N $AgNO_3$	Cl mN	Neutral Cl mN
8.25	10.0	0.0	4.35	0.25	3	0.81	8	5

able that a corresponding amount of water was resorbed. Therefore, when calculating the primary acidity in this case, I have added 0.2 ml to the measured volume increase.

At 8.30 a. m. 0.11 mg histamine hydrochloride was administered subcutaneously. 10 ml glycocoll solution was flushed down every 15 minutes beginning at 8.45 a. m. and, after aspiration, the following values were obtained (tab. 2).

Table 2.

Time	Amount ml	Increase ml	pH	H mN	Cl mN	NeutralCl mN	Primary Acidity mN	Remarks
9.00	12.5	2.5	3.5	15	21	6	68	Restless
9.15	10.8	0.8	3.45	14	20	6	155	Slept

Case 2. Age 7 weeks. Weight 3930 g. Examination carried out similarly to above. The 1st 10 ml glycocol solution passed down at 7.55 a. m. Water resorption estimated at 0.1 ml in 15 minutes. The following table (3) shows result.

Table 3.

Time	Amount ml	Increase ml	pH	H mN	Cl mN	Neutral Cl mN	Primary Acidity mN	Remarks
8.10	10.8	0.8	4.05	6	15	9	76	Restless
8.25	10.8	0.8	3.65	11	15	4	131	Quiet
8.25	subcutaneous injection of 0.055 mg Histamine hydrochlor:							
8.40	10.3	0.3	3.95	7	15	8	185	Quiet
8.55	10.9	0.9	3.80	10	18	8	103	Quiet
9.10	10.9	0.9	4.25	5	10	5	51	Quiet

Result. The values of the primary acidity obtained vary considerably. If one disregards the figure 185, which, having regard to the measure accuracy ± 0.1 ml, is very uncertain in so small an increase in volume as 0.3 ml, we have remaining the figures 68, 155, 76, 131 and 103 mN. The margin of discrepancy for these figures is ± 15 % by the increase in volume of 0.8 ml and decreases with higher increases in volume.

The majority of the acidity values stated above are higher than that which is usually given as the acid amount in children of that age and therefore they give support to the supposition of high primary acidity even in very small children.

The methods used have not permitted an absolutely exact examinations on children. I have therefore completed them with experiments on kittens.

Experimental investigations on kittens.

Examinations of primary acidity and diffusion conditions in the ventricles of grown-up cats have been previously published by Teorell (1939, 1940). The results achieved by him have therefore been a valuable material of comparison for me.

My idea was to examine kittens of an age which could compare with a child of not more than six months. Naturally it is impossible to say with any degree of certainty that a certain cat-age corresponds with a certain human-age.

But on the ground that the lactation time of a cat is about 5 weeks whilst that of a human being is between 7 and 9 months, one can calculate that a 3 weeks old kitten corresponds approximately to an infant of 4—5 month. The following examinations were made on kittens of 3 weeks of age.

Primary acidity in kittens.

Procedure. The experiments were carried out in the morning after the cat had been fasting for at least 12 hours. Ana-

esthetic: Ether + chloralose-urethane intravenously. Laparotomy Subligation of the oesophagus at the cardia as well as of the pylorus. All ligatures in subligation were placed under the visable blood vessels so that they should not be constricted. This avoided nutrition damage to the stomach. Further it did not prevent the histamine introduced into the blood stream from reaching the mucosa. Through an opening in the wall of the stomach at the pylorus a small glass canula was placed and fixed with a ligature. The peritoneal cavity was closed round the free end of the canula. Through this a 0.9 % NaCl solution was passed and the canula closed with a rubber stopper. After this there was a pause for at least 30 minutes.

Method of examination. The examinations were carried out mainly in the same manner as already described for children. The buffer solution is aspirated through a fine rubber tube placed in the canula. Accuracy of measure was ± 0.1 ml.

An example of the results of the examination: In order to obtain sufficiently large amounts of gastric juice from these small animals, the experiments were made in periods of 1 hour. A couple of typical cases will be given here.

Table 4.

Time	Amount ml	Increase ml	pH	H mN	Cl mN	Neutral Cl mN	Primary Acidity mN	Histamine Hydrochlor. Subcutant mg
12.00	3.4	0.4	2.10	42	52	10	240	
13.00	3.9	0.9	2.15	70	81	11	250	
13.00								1.1
14.00	3.95	0.95	2.35	68	76	8	234	
14.00								1.1
15.00	4.1	1.1	2.20	78	88	10	247	
15.00								1.6
16.00	4.4	1.4	2.30	78	87	9	214	
16.00								1.6
17.00	4.1	1.1	2.35	66	76	10	209	

Cat No. 12. Weight 230 g. When calculating the primary acidity the measured volume increase is corrected with + 0.2 ml (= experimental recognised water resorption). At 11 a. m. a subcutaneous injection of 0.8 mg Histamine hydrochloride was given and subsequently in doses and at the times stated in table 4 below. At 11 o'c 3.0 ml glycocol solution (pH 6.0) was passed down. The ventricle was emptied every hour afterwards and fresh 3 ml quantities of glycocol solution passed down. The examination of the aspirated solution showed the results above (tab. 4).

Cat No. 9. Weight 330 g. The following values for primary acidity were obtained from this cat: 205, 212, 154, 173 mN. Volume increase per hour varied between 1.6 and 3.8 ml.

For comparison with these primary acidity values, an estimation for secondary acidity was made on cat No. 9.

After the ventricle had been aspirated empty a subcutaneous injection of 0.55 mg histamine hydrochloride was given at 22.00 p. m. The whole gastric juice was aspirated every 20 minutes and examined with the following result.

Table 5.

Time	Amount ml	H (pH 3.5) mN[1]	H (pH 7.4) mN	Cl mN	Neutral Cl mN
20.20	1.9	125	137	163	26
20.40	1.3	127	142	165	23
21.00	1.0	120	134	162	28

[1] Titration to pH 3.5 and 7.4 with indicators.

Summary.

Primary acidity in infants and kittens.

If one includes all primary acidity values where the volume increase has been 1.0 ml or over (which signifies an error in value of at the most ± 10 %), one arrives at the following figures: 247. 214, 209, 205, 212, 154, 173 and 254 (cat No. 11). The average will be 209 ± 12 mN. This coincides well with TEORELL's value (208 ± 6 mN) for grown-up cats.

Regarding this and supported by the direct examination of primary acidity in children there are good grounds for supposing that the primary acidity of the gastric juice even in children is just as high as in adults.

The above comparisons between kittens and children are made with all the reservation with which one should judge the result of animal experiments when putting it into practice on man.

Electrolyte Diffusion in the Stomach Mucosa.

It has been shown in the previous chapter that gastric juice has a high, almost constant acidity at the moment of secretion even in young people. This primary acidity falls quickly to a lower value, secondary acidity, depending on the influence of acid-lowering factors such as the admixture of saliva, mucus and food as well as regurgitation of alkaline duodenal contents.

Teorell (1933, 1935) has, however, shown by experimental examinations on grown-up cats that the fundamental part of acidity regulation takes place as a result of a diffusion of the H-ions of gastric juice through the stomach mucosa in exchange for alkali-ions from the blood.

The acidity decrement, which in man can be calculated to happen through exchange diffusion, has been calculated by Elliot, Risholm and Öbrink (1942) as being 50—80 % of the total acidity regulation for adults.

The possibilities for examining the exchange diffusion in the human ventricle are limited on account of the difficulties in procedure. Under good experimental conditions, however, I have been able to carry out a similar examination on a five-day old child and can therefore endorse Teorell's theory about the importance of the exchange diffusion's acidity decrement in man.

Teorell (1939) has investigated the diffusion in the ventricle of grown-up cats for different electrolytes. My investigations have been carrid out on kittens of about 3 weeks old.

Procedure.

The diffusion experiments were made with an approximately 170 mN HCl of which a measured quantity was passed down into the ventricle through a small canula such as is described above. Every 15th minute the whole amount was aspirated and 0.1 ml removed for examination whilst the remainder was again passed down into the ventricle. The portion removed for testing was so small a quantity as 0.1 ml in order that the total amount of solution placed in the stomach should decrease as little as possible.

In certain cases the diffusion speed was also tested with 1 % ammonium nitrate with which not only the outward diffusion of NH_4, but also the inward diffusion of Cl to the stomach could be studied.

Determination of Acidity: 0.1 ml HCl solution was titrated with 0.01 ml carbon dioxide free NaOH (bromthymol blue as indicator) to a similarity in colour with a buffer solution of pH 7.4. Degree of accuracy was $\pm$ 1 mN.

In order to correct faults in titrating and pipetting, 0.1 ml of a 0.100-N HCl solution taken with the same pipette was also titrated with the above 0.01-N NaOH, after which the acidity value in the sample was corrected.

Determination of chloride: The same sample which previously had been used for the determination of acidity was titrated electrometrically with 0.005-N $AgNO_3$ after it had been made acid with 1 ml 15 % trichlor acetic acid. Degree of accuracy $\pm$ 1 mN.

Determination of Ammonium: The ammonium content was determined in PULFRICH's spectrophotometer after the 1 % sample quantity (0.1 ml) of ammonium nitrate had been nesslerized and diluted with aq. dest. (1: 25).

The extinction of the clear yellow colour, which had thus arisen, was determined in spectra filter S 43 where the extinction was directly proportional to the ammonium concentration. The same dilution (1: 25) of the original 1 % solution (125 mN) was used as a standard solution. Degree of accuracy was $\pm$ 2 mN.

Diffusion experiments on Children.

A diffusion experiment, under favourable conditions, was made on a 5-day old child with a serious deformity (atresia oesophagi + fistula tracheo-bronchialis). In connection with the establishing of a ventricle fistula a temporary closing of the pylorus was made. Thereupon two diffusion trials were

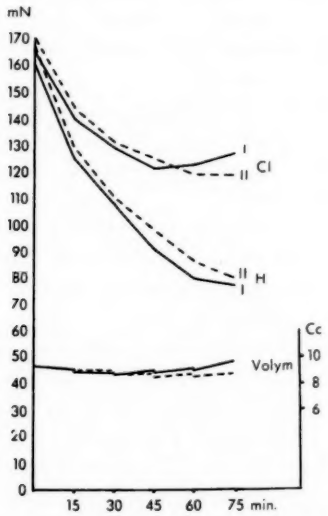


Fig. 1. Change in concentration and volume of a HCl solution which has been passed down into the ventricle. (Cc = ml).

made; one of them (I) with 165 mN HCl and the other (II) with 173 mN HCl (see fig. 1).

Result. Acidity dropped rapidly whereas the chloride content fell more slowly and showed tendencies to remain stationary. Coinciding results were obtained in both experiments. Admixture with saliva or duodenal content was, in accordance with the experimental conditions, excluded.

Prior to the experiment the ventricle content was Kongo -neg.

As far as I know this is the only published diffusion experiment on man where accurate determinations of volume and concentration could be carried out on the total amount of the solution placed in the ventricle.

The result supports Teorell's theory that decreasing of the acidity of the gastric juice by exchange diffusion is also suitable for man.

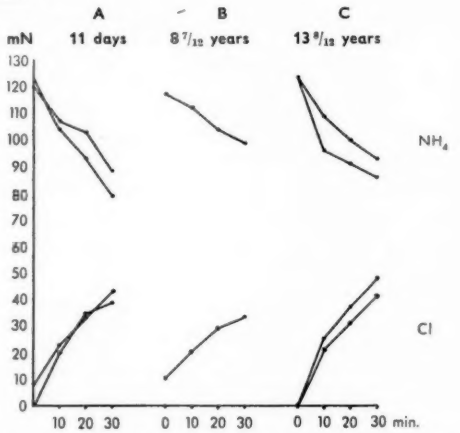


Fig. 2. Diffusion experiments with 1 % ammonium nitrate on three children of different ages.

An experiment in the comparison of diffusion speed in children of different ages was also made. Into the ventricles of three children aged 11 days, 8 7/12 and 13 8/12 years respectively 10 ml, 49 ml and 65 ml 1 % solution NH_4NO_3 was introduced. These quantities were measured out in proportion to the calculated stomach surface of each child.

Two experiments were made on the eldest and youngest child. The ventricel content during the experiment was Kongo -neg. The current changes in concentration from out-diffusion of NH_4 and in-diffusion of Cl will be seen from Fig. 2.

From the above curves it will be seen that the diffusion condition for these children of different ages were on the whole similar under the said experimental circumstances.

Diffusion experiments on kittens.

TEORELL (1939) in his work »On the permeability of the stomach mucosa for acids and some other substances» showed how the concentration of, for instance, 10 ml 170 mN HCl changed whilst they were introduced into the stomach of a grown-up cat for 75 minutes. The curve to the extreme left in Fig. 3 below refers to this experiment of TEORELL. In order to compare TEORELL's experiment on grown-up cats I performed the following trial diffusions on kittens.

Procedure: The ventricle was ligated at the oesophagus and pylorus and a glass canula was operated into the wall of the ventricle as previously described.

On aspiration the accuracy of the fluid from the ventricle was $\pm$ 0.1 ml. Removal of the sample was carried out in the same way as in diffusion attempts on children. The amount

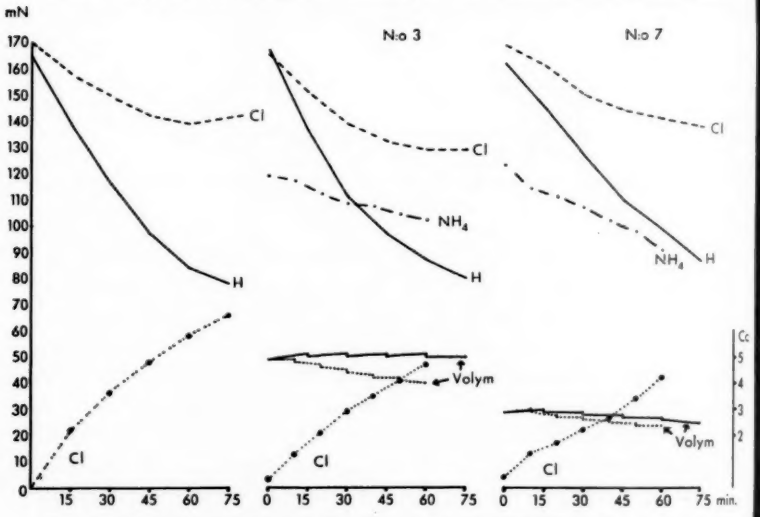


Fig. 3. Comparison between diffusion speed for 170 mN HCl in grown-up cat (left curve) and kittens (Nos. 3 & 7). ——— = acidity (pH 7.4) and - - - - - = chloride in experiment with 170 mN HCl.

NH_4 (–·–) and Cl (●··●) represents diffusion trials with 1 % ammonium nitrate. ⎍⎍ = ml HCl, ⎍⎍ = NH_4NO_3. (Cc = ml).

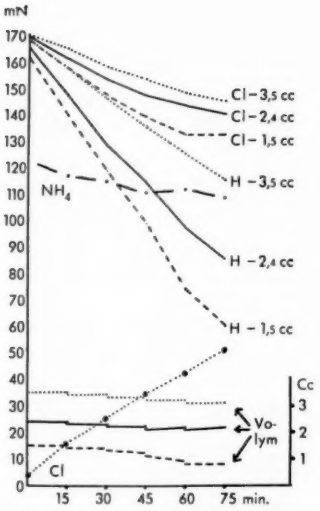


Fig. 4. Cat No. 8. Diffusion experiment with different amounts (1.5, 2.4, 3.5 ml) of 170 mN HCl placed in the ventricle. (NH_4 (–·–) and Cl (●····●) represents the diffusion experiment with 1 % ammonium nitrate. (Cc = ml).

of diffusion fluid was selected in accordance with the calculated surface of the animal's stomach. The diffusion was examined for 170 mN HCl as well as for 1 % ammonium nitrate.

The first examination showed the distinct acidity decrement as well as, in the beginning, a moderately reduced and, later on, a relatively constant Cl content. The latter showed, not only a decrease in NH_4 concentration but also an increase in the indiffused Cl from the blood of the mucosa to the fluid.

Experimental animals: Cat No. 3. Weight 600 g. Age 6 weeks. Cat No. 7. Weight 200 g. Age 4 weeks.

Result. From fig. 3 scarcely any difference between the diffusions in the three cats of different ages will be seen.

Importance of the volume of the diffusing fluid. The speed of change in the concentration of the solution in the ventricle is dependent, amongst other factors, on the volume of the solution. This is apparent from an experiment on one and the same cat with different diffusing fluids (cat No. 8. Weight

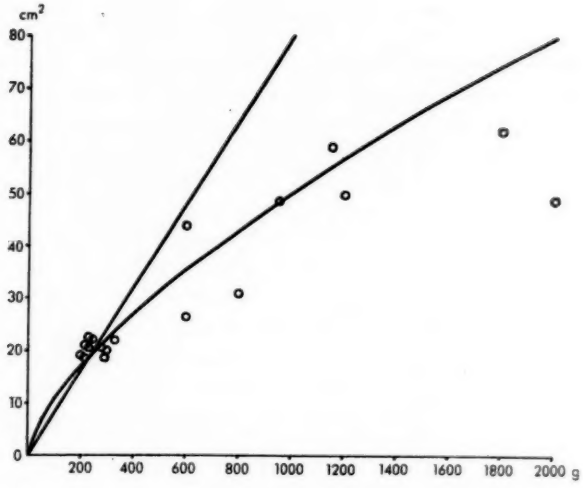


Fig. 5. The curve showing size of measure of stomach surface in cm² (O in different sized kittens. The lines give the calculated size of stomach surface (with base of 250 g body weight and 20 cm² stomach surface) partly in relation to the body weight according to lineal proportion (right line) and partly in relation to the body surface according to formula $k\sqrt[3]{kg^2}$ (bent line) . [g = k · body weight in gram.]

200 g. Age 3 weeks). The amounts of mN HCl and the decrement in concentration are shown in fig. 4. The amount of ammonium nitrate was 2.4 ml.

On the assumption that a grown-up cat with a supposed weight of about 3 000 gm gets 10 ml, cat No. 8 ought to have received 1 ml if the amount of the solution had been reduced in proportion to the body weight, but 2.2 ml in proportion to the surface of the body (and surface of the stomach. See below).

When making comparative examinations for different ages I considered it suitable to use a volume of diffusion fluid which was proportional to the stomach surface. That which agreed best with the results of the diffusion on grown-up cats (compare fig. 3) was also obtained in this experiment

where the volume of the diffusion fluid (2.4 ml) was measured approximately in proportion to the surface of the stomach.

The surface of the stomach. An examination of the surface of the stomach of cats of different ages shows that it is proportional to the surface of the body and not to the weight of the body which is shown from the curve (fig. 5), based on direct measurement of the surface of the stomach of 18 cats immediately after the death.

Summary of diffusion experiments on kittens.

When comparing the examinations between kittens and grown-up cats it was not possible, in the case of the young animals, to prove any increase in the lowering of acidity through increased diffusion in those cases where the amount of liquid passed down was measured in proportion to the surface of the stomach.

Diffusion in different parts of the ventricle.

Having regard to the different histological structures of the mucosa, it seems interesting to me to study the diffusion in the different parts of the ventricle. This is divided in the middle by a ligature so that there is no connection between the upper and lower parts.

The diffusion is studied partly in both parts simultaneously and partly separately.

The experiment did not give any definite evidence that diffusion took place at different speed in any part of the ventricle.

Diffusion before and after atropine medication.

Spontaneous secretion of HCl and mucus can be thought to influence acidity changes in the diffusion experiments referred to above. I therefore thought it also suitable to examine the diffusion after strong medication with atropine. By this means both HCl and mucus secretion could be suppressed.

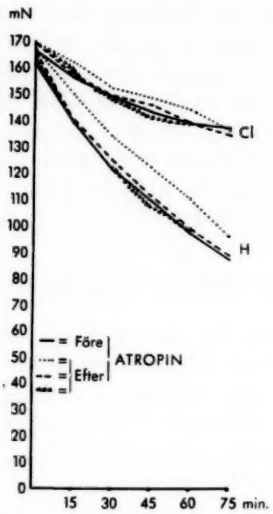


Fig. 6. Cat No. 14. Diffusion experiments with 170 mN HCl before and after atropine medication. (»Före» = before; »Efter» = after.)

Fig. 6 shows an experiment with 170 mN HCl which was made on a kitten (No. 14) 3 weeks old weighing 240 g. An experiment was first made without atropine after which a total of 3 mg atropine sulphate was given before the following three experiments (fig. 6).

The diffusion, tested for H and Cl, proceeded in the same manner both before and after atropine medication. This fact can be of interest when thinking about atropine medication in, for instance, hyperacidity.

Conclusions.

1. The primary acidity of the gastric juice proves to be high even in infants although as equally high a value has not been shown as in adults. Apparently this depends on technical difficulties.

2. The primary and secondary acidity of the gastric juice

in kittens shows the same value as in grown-up cats. This supports the supposition that on the whole the same acidity conditions apply also to man in all ages.

3. Comparative examinations of the diffusion conditions in the ventricle of younger and older children as well as in kittens of different ages show similar diffusion for all ages provided the fluid amounts are weighed in proportion to the surface of the stomach.

4. Atropine has no influence on the course of the diffusion and there are no marked differences in diffusion between the upper and lower parts of the ventricle.

PART II.

Clinical Studies of the Secretion and Composition of Gastric Juice in Healthy Children.

Previous investigations.

For more than fifty years there have been a profuse number of publications concerning the investigation of gastric juice in children. In order to make a closer study regarding the development of the methods of investigation the following publications, giving good surveys, are recommended: Ad. H. Meyer (1902), Hess (1913), Demuth (1922), Muhl (1925), Izmuita (1930), Bent Andersen (1935), Huhtikangas (1936), Kaijser (1943) and Flensborg (1944). It may especially be referred to the work by Flensborg, because it deals with investigations very similar to my own.

Summarily it can be said that one had found that the stomach of the new-born infant contains free hydrochloric acid but with a low acid content and that this rises with the age of the child. One considered oneself entitled to raise the so-called normal value for acidity in different ages. Too great a variation in these values by the different authors in which the difference in the amount and kind of test meal played a part, made them clinically less useable.

Therefore one began to examine as pure a gastric juice as possible and passed over from test meals to other stimulants such as alcohol, coffeine or histamine.

The first person to use histamine in gastric juice examinations in children appears to be Harmelin (1925). Investigations with histamine stimulation in children have been published by Coari (1927), Neale (1930), Diedrich and Shelby

(1931), Siemsen (1932), Lehmann (1934), Faber, Mermond, Gleason and Watkins (1935), Cutter (1938), Kreutzfeldt and Flensborg (1942).

Since examinations have been carried out by means of histamine, one got knowledge that even children could have a high HCl value. Kaijser (1943) and Flensborg (1944) could show that the primary HCl acidity is probably just as high in all ages but that technical details make it difficult to give direct proof of this in the youngest ages.

According to Flensborg the most probable reason for the appearance of the lower secondary HCl values in the youngest ages seems to be an increased diffusion in the stomach walls.

By continually aspirating saliva, duodenal and gastric juice separately, Ihre (1938) was able to show in adults that the normal state for a healthy person's gastric juice was the presence of a high acidity with an almost constant maximum.

Gastric juice secretion following parasympathetic stimulation with, for instance, insulin has been studied by Detre and Sivé (1924), Roholm (1930), Kalk and Meyer (1932), Ihre (1938) and Horstmann (1939).

From this one has usually found that gastric juice, after insulin stimulation, has a higher pepsin content and more often a higher acidity and greater volume than after histamine stimulation.

I have not been able to find anything written before 1943 about gastric juice examinations on children after stimulation with insulin and therefore considered it valuable to make an experiment.

The chloride content of gastric juice has been investigated by Török and Kellner (1924), Copeman and Hill (1928), Stokes, Stephenson and Garret (1929), Bent Andersen (1935) and others. No coincidental values have been obtained.

Previous examinations of the gastric juice content of pepsin have mainly been of a qualitative character and it has been a question of whether children with their low acidity could really possess an active pepsin.

Examinations for the pepsin activity in gastric juice are

more valuable as good quantitative methods of examination are obtained. Such a method was worked out by LINDERSTRØM-LANG in 1931. BENT ANDERSEN (1935) investigated pepsin activity in gastric juice by this method and showed that it increased in proportion to the child's advancing age.

ANDERSEN made an examination of a gastric juice which had been diluted twenty times and which had been obtained after a test meal.

IHRE (1938) determined the pepsin content of gastric juice in adults after stimulation with histamine and insulin. The highest pepsin content was obtained after insulin stimulation.

Queries.

A perusal of literature shows that amongst other things the following questions are less well explained.

Are the published values of the acidity of the stomach content (secondary acidity) a proper expression for the HCl producing power of the stomach in children of different ages and is it accordingly permissable to talk about rising normal values for HCl according to rising age?

Can the variation in the published so-called normal HCl values for the stomach content be thought to depend on deficiency in the methods used for examination?

If that is so, can one, by means of an improved method, prove the same secondary acidity and chloride content in the ventricle in children of different ages as in adults?

Can one, by examining the purest possible gastric juice, even for pepsin, prove the same concentration on the whole for all ages?

What is the most suitable method of making a gastric juice examination on children with histamine or insulin stimulation?

What diagnostic advantages can a histamine or insulin test on children offer in comparison with other kinds of gastric juice examinations?

Personal investigations.

In order, as an answer to the above questions, to try and unravel gastric juice conditions in children in comparison with those in adults, the examination should be carried out by the same method for the whole scale of ages from infancy to adult. The following is an account of such an investigation.

Examination cases. My investigations were carried out on healthy children. The classification of children according to age and sex is shown in the following table (6).

Table 6. Classification of normal cases according to age and sex.

Group	Age, years	No. of cases	Boys	Girls
I	14 $^{11}/_{12}$ to 10	10	4	6
II	9 $^{11}/_{12}$ to 5	10	6	4
III	4 $^{11}/_{12}$ to 1	11	3	8
IV	$^{11}/_{12}$ to $^{6}/_{12}$	9	6	3
V	$^{5}/_{12}$ to new born	5	2	3
	Total	45	21	24

On the whole the methods of examination used agreed with that described by Ihre (1938) for adults. The following standards for examination were put up.

1. It should be made preferably on a lesser number of children and as technically complete as the circumstances in the younger permits, than in a more simple manner on a larger number of children.

2. It ought to be done after the same method of complete age series (from newly born to fifteen years) which would ensure a better comparison of results.

3. It ought to be done as thoroughly as possible for which reason the determination of gastric juice volume, the acidity, chloride and pepsin content must be carried out after both histamine and insulin stimulation.

Great patience is demanded of the examiner if he adheres to these conditions in a children's practice. One hundred cases had to be examined in order to obtain a useable material concerning some fifty odd children with normal stomachs.

The examination began round about 8 o'c in the morning and lasted for about three hours. The children had been fasting from 18.00 o'c the previous evening.

The children had to lie on a bench, the head end of which was raised 10 degrees horisontally. Saliva was continuously aspirated.

Narcosis. Despite the aspiration of saliva it was not possible to prevent saliva or mucus from passing down into the ventricle of restless or small children. This caused a dilution of the gastric juice and a lowering of acidity which was considerable in the smaller children in whom the volume of gastric juice was small.

I therefore gave the smallest children a chloral narcotic. The importance of this, having regard to the reliability of results obtained, can be seen from fig. 14.

The rather large dose of chloral was given in accordance with the following table (7).

Table 7. In rectal narcosis use chloral in doses proportionate to the body weight of the child.

Body weight of child kg	Gramm Chloral approximately
— 4.9	0.5
5— 7.9	0.75
8— 9.9	1.0—1.4
10—11.9	1.5—1.9
12—15	2.0

Stomach tube. Taking the height of the child as a basis, the following lengths of tubing are used calculated from lower teeth (fig. 7).

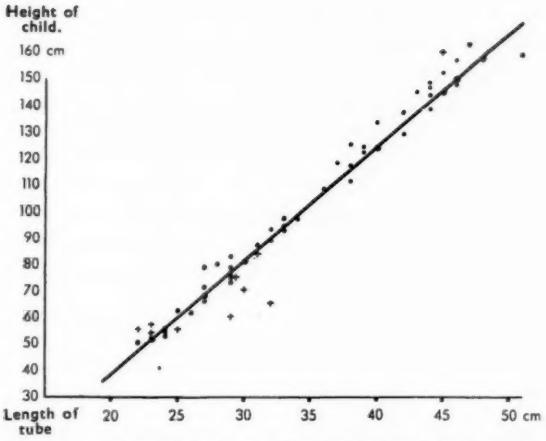


Fig. 7. The curve shows length of tube in relation to height. (● = length of tube used in each examination. + = post mortem control in certain cases.)

A receptable is fastened to the upper end of the stomach tube to which a suction apparatus with a pressure of 25 mm Hg is attached for aspiration of gastric juice continuously during the test period. This enables even small quantities to be collected without loss.

To begin with, the content of the fasting stomach together with the spontaneous secretion is aspirated for a period of 20 minutes. A subcutaneous injection of histamine was then given and the gastric juice collected fractionally during the following three or four 20-minute periods. When the effect of the histamine could be thought to have ended, a dose of insulin was injected intravenously as a stimulant for fresh gastric juice secretion. Then, for at least one hour more, the gastric juice was collected every twenty minutes.

Ihre (1938) resorted to simultaneous aspiration from the ventricle and duodenum during his experiments in order to prevent regurgitation of duodenal juice. I used a similar method during a number of preparatory tests but found that the gain was outweighed by the extra trouble. An admixture of bile with the gastric juice very rarely happened with the chil-

dren for which reason the above more simple method gave sufficiently good results with less trouble.

Histamine as a stomach stimulant in children.

Histamon (Messrs Tika Ltd. Sweden) which contains 0.55 mg histamine hydrochloride per 1 ml was used as a stimulant. It was administered subcutaneously.

Visible increase in gastric juice secretion took place usually after 20—30 minutes and reached its maximum 40—60 minutes after injection.

About 150 children varying in age from newly born to 15 years were examined without suffering any ill effects from histamine.

Histamine has been administered in doses as per table 8 below calculated in proportion to the body surface on the basis that a child weighing 50 kg should receive a dose of 0.55 mg.

Body surface (S) has been calculated after formula $S = K\sqrt[3]{kg^2}$; K = constant factor, kg = body weight in kg.

Table 8.

Body weight of child kg	Histamine Chloride mg
3	0.08
5	0.12
7	0.15
10	0.18
15	0.24
20	0.30
25	0.35
30	0.40
35	0.44
40	0.47
45	0.51
50	0.55

In certain preliminary examinations I found that the action of histamine was less certain on the smaller children if the dose was administered in proportion to body weight.

Flensborg (1944) considers that a sufficient histamine effect can be obtained from doses of 0.01 mg histamine per kg body weight for all ages. This is also the dosage which is usually used for adults.

Personally I strived to obtain a strong effect of histamine in all cases. It was first when I altered the dosage in conformity with the above table that I considered that I had got a sufficiently strong effect.

Insulin doses.

In the same manner as Ihre (1938) I also used insulin which is a powerful stimulant for pepsin secretion. Insulin was given intravenously to the bigger children and intramuscularly to the smaller ones. The dosage, calculated in accordance with their body weight, will be seen from Tab. 9 below. (IU = International units).

Table 9.

Body weight of child kg	Insulin IU	Insulin ml
3.0	2	0.05
6.8	4	0.10
11.4	6	0.15
17.6	8	0.20
25.2	10	0.25
33.4	12	0.30
41.6	14	0.35
50.0	16	0.40

Marked increase in gastric juice secretion was not often encountered until 40—60 minutes after injection of insulin.

Examination of gastric juice after test meal.

In almost every case an EWALD's test meal was given as a control. The test meal given to children up to 2—3 years of age consisted of 15 g rusks and 150 g water. The gastric juice was titrated with TÖPFER's reagent on free HCl and with phenolphthalein for the total acidity.

Chemical analytical procedure.

1. Acidity.

Acidity is expressed in millinormals (mN). In general the method recommended by TEORELL (1933) was used. 0.1 ml clear gastric juice + 0.25 ml carbon-free aq. dest. + 1 drop bromphenol blue was titrated with 0.01-N NaOH to uniformity in colour with a buffer solution of pH 3.5. Titration to this pH was taken as the measure for free HCl.

One drop of bromthymol blue was added to the same test and afterwards titrated with 0.01-N NaOH to a uniformity in colour with a buffer solution of pH 7.4.

The total amount of NaOH hereby consumed made the measure for total acidity. Degree of accuracy when titrated was $\pm$ 1 mN.

2. Chlorides.

These were determined by the same test which was used for determination of acidity. Titration was carried out electrometrically (see TEORELL 1933) with 0.005-N $AgNO_3$. Chloride content was expressed in mN. Degree of accuracy during titration was $\pm$ 1 mN.

3. Pepsin.

The analysis was made chiefly in accordance with a micro method described by LINDERSTRØM-LANG 1931. An extract of albumen was digested at pH 2.1 with pure gastric juice diluted to 1: 10. The digested mixture always made 0.15-N having regard to Cl. The pH adjustment was made electrometrically with antimony electrodes.

Substratum. A solution of 4 g edestin in 100 ml 0.056-N HCl was used as a substrate. This solution was corrected to pH 2.1 by an insignificant amount of HCl.

Digestion method. By means of a 8.05 mm^3 pipette (the same one was used during the complete series of tests) a solution of edestin and an equal quantity of the above diluted gastric juice was passed into the bottom of a 250 mm^3 micro test tube of Jena glass. In the control test the drop of edestin is placed on the wall of the micro test tube and not at the bottom of it. Digestion was carried on for half an hour at 40°. After half an hour about 150 mm^3 acetone was added to the digestion and control test by which means the digestion was stopped. Acetone contained 15 % aq. dest. with naphthyl red as an indicator.

Titration. Titration was then carried out with 0.05-N HCl in 95 % alcohol and in accordance with LINDERSTRØM-LANG.

Pepsin content. LINDERSTRØM-LANG, HOLTER, BENT ANDERSEN and others have shown that if a certain amount of gastric juice with its enzyme produces a certain degree of splitting of albumen, a double quantity of enzyme will not produce twice the amount of splitting during the same time. The relation between enzyme content and digestion degree shows a proportionality which can be expressed in accordance with an empirically obtained curve. The curve below (Fig. 8) gives the digestion degree expressed as the amount of titratable split-products (= mm^3 0.05-N HCl), in different dilutions (1: 40, 1: 10, 1: 8 etc.) of the same gastric juice during the same digestion time.

One can then propose that a certain degree of digestion (in this case 2 mm^3) shall signify the presence of a certain amount of pepsin units (in this case 0.01). Double the amount of gastric juice must then contain twice as many (0.02) pepsin units etc. One can therefore get a direct measure from the curve for mm^3 HCl consumed during titration expressed in pepsin units (PE).

Standard Curve. The gastric juice was taken from a 9-year old child as a working basis. Digestion was carried out on 7

different dilutions and according to the above description. The following curve was obtained.

Control of the correctness of the appearance of the standard curve was carried out with a mixture of five different gastric juices. The values obtained from this experiment are marked with a cross on the standard curve (fig. 8).

As the dilution and test volume of the diluted gastric juice in digestion was always known, the number of consumed

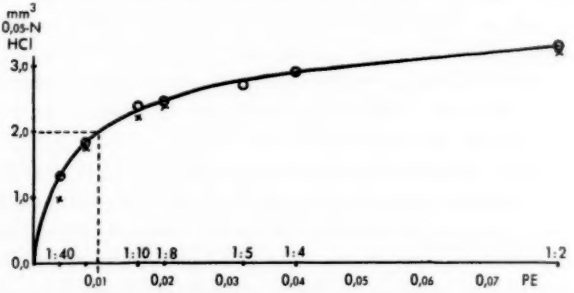


Fig. 8. Standard curve for pepsin units (PE). x-value represents result of a control test. Gastric juice dilution is shown by 1: 40 etc.

mm^3 HCl could be expressed directly in PE per mm undiluted gastric juice.

The calculation of the capacity of the micro pipette was made by 200 titrations with 0.100-N HCl. The average was 8.05 mm^3.

Digestion experiments with crystalline pepsin. In order to fix the pepsin units used by me in order of size, a digestion was made with crystalline pepsin. The basis was pepsin WITTE and the preparation was made according to PHILPOT and SMALL (1938) with modifications according to instructions from Mr. G. ÅGREN, M. D., Uppsala.

The crystalline pepsin was dissolved in a dilution of 1:10 000 in 0.9 % NaCl and set to pH 2.1. The digestion was made with 8.05 mm^3 of this dilution.

1 g of this crystalline pepsin proved to correspond with 17 400 PE (the pepsin-unit used here).

For comparison with the pepsin figures in adults arrived at by IHRE (1938), it can be stated that 1 g of the crystalline pepsin examined by him corresponded with 34 600 of his pepsin units.

For all the digestion experiments with gastric juice in this work I make the proviso that by pepsin content is meant the capacity of the gastric juice to digest albumen substrate without any further analysis being made to ensure that the action of enzyme just depends on the presence of pepsin only. As the digestion was made at pH 2.1, the presence of pepsin is considered most probable.

Examination results.

Table 10. Group I. Children in ages 14 $^{11}/_{12}$ to 10 years without any known disease of the stomach.

	No.	1	2	3	4	5	6	7	8	9	10
	Sex	♀	♂	♀	♂	♂	♀	♀	♂	♀	♀
	Age in years	14 $^{2}/_{12}$	13 $^{2}/_{12}$	13	12 $^{2}/_{12}$	12 $^{2}/_{12}$	11 $^{10}/_{12}$	11 $^{2}/_{12}$	10 $^{9}/_{12}$	10 $^{7}/_{12}$	10 $^{3}/_{12}$
Histamine test	Amt/60 min	105	49	109	136	105	109	98	107	79	113
	Max amt/20 min	39	20	43	56	39	51	39	37	54	41
	Max free HCl	137	108	132	132	110	114	111	106	104	139
	Max Ta	147	119	142	143	119	124	119	116	113	150
	Max chloride	166	157	163	162	142	157	151	149	145	169
	Max PE/ml	22	25	25	14	25	8	29	6	17	16
	Max PE/20 min	800	500	1070	550	920	350	830	190	670	540
	Amt PE/60 min	1580	770	1630	1580	1900	720	1950	390	1070	1520
Insulin test	Max amt/20 min	18	12	19	27	52	45	15	12	41	23
	Max free HCl	124	86	109	123	128	123	90	98	85	111
	Max Ta	135	98	121	135	137	134	100	108	92	126
	Max chloride	159	145	151	155	158	161	147	151	146	162
	Max PE/ml	25	14	9	19	50	14	62	29	29	62
	Max PE/20 min	430	160	120	420	840	640	930	580	1200	1300
	Free HCl EWALD	36	40	25	37	30	38	30	15	5 6	2

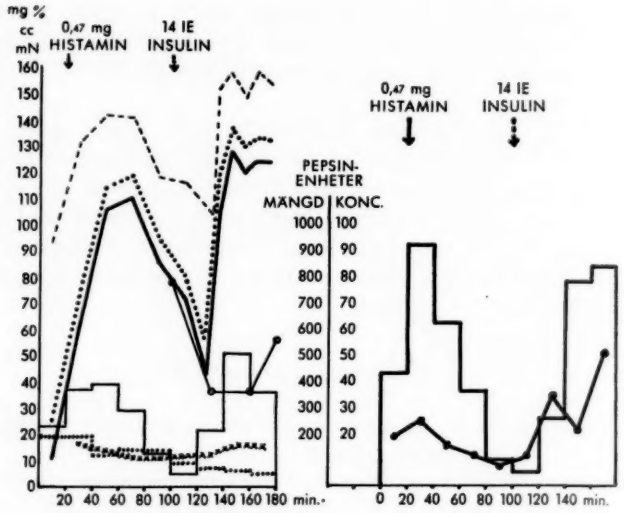


Fig. 9. Case No. 5. Age 12 $^{9}/_{12}$ years. Graphic presentation of histamine and insulin tests. For the sake of lucidity the diagram is put up in two parts each representing the same child and the same time of examination.

Left part. ----- = total chloride
——— = free HCl
. . . . . = total acidity
xxxxx = saliva chloride
——— = amount of gastric fluid.
. . . . . = amount of saliva
O—O = blood sugar mg %

Right part. ——— = Pepsin Units (PE) number
●—● = Pepsin Units (PE) concentration
(»Pepsinenheter» = Pepsin units; »Mängd» = number

A synopsis of the results is given in the tables and diagrams below. The following abbreviations have been used: amt/60 min = total amount of gastric juice in 60 minutes: max amt/20 min = the maximum amount in 20 minutes, both expressed in ml. Max. free HCl, Ta and chloride = maximum concentration of free HCl, total acidity and chloride in a 20 minute test expressed in mN. Max PE/ml = the maximum concentration of pepsin units per ml in a 20 minute test, and max. PE/20 min = the maximum total number of PE in a

Table 11. Group II. Children in ages 9 $^{11}/_{12}$ to 5 years without any known disease of the stomach.

No.		11[1]	11[1]	12	13	14	15	16	17	18	19	20
Sex		♂	♂	♂	♂	♀	♂	♂	♀	♀	♀	♂
Age in years		9 $^{8}/_{12}$	9 $^{8}/_{12}$	9 $^{7}/_{12}$	9 $^{6}/_{12}$	9 $^{1}/_{12}$	8 $^{9}/_{12}$	8 $^{7}/_{12}$	7 $^{9}/_{12}$	7 $^{1}/_{12}$	6 $^{10}/_{12}$	6 $^{2}/_{12}$
Histamine test	Amt/60 min	82	63	77	66	55	55	57	64	76	53	32
	Max amt/20 min	30	22	37	51	22	19	24	24	38	23	17
	Max free HCl	128	128	122	104	123	114	124	98	106	119	111
	Max Ta	140	139	135	116	132	127	142	108	122	130	122
	Max chloride	166	160	160	151	155	156	157	145	152	156	151
	Max PE/ml			22	29	12	22	8	8	12	14	10
	Max PE/20 min			410	1490	260	400	130	160	320	300	160
	Amt PE/60 min			1040	1820	530	1000	380	460	520	560	300
Insulin test	Max amt 20/min	24	11	25	23	27	29	15	17	3	20	22
	Max free HCl	124	93	97	115	123	120	116	66	13	121	129
	Max Ta	137	105	109	128	133	134	133	74	24	132	140
	Max chloride	163	157	154	152	155	158	157	143	116	158	153
	Max PE/ml			22	29	22	41	12	16	8	17	20
	Max PE/20 min			460	300	220	860	150	270	20	260	440
Free HCl EWALD		13		5	27	18	2	7	28	40	15	17

[1] Same patient on two different occasions.

similar period. Free HCl EWALD = acidity titrated to pH 3.5 in the test meal.

Group I. No narcosis was used in any one of these cases and the examinations caused no great trouble.

As an example of the graphic presentation of the tests, the following case, No. 5 (fig. 9) has been chosen for this group.

Group II. Avertin narcosis was used in case No. 20. Case No. 15 (fig. 10) is used as an example of the graphic presentation of this group.

Group III. In cases 22 and 28 the examination could be carried out without narcosis. It shows that even in children of this age one can carry out a histamine test without narcosis.

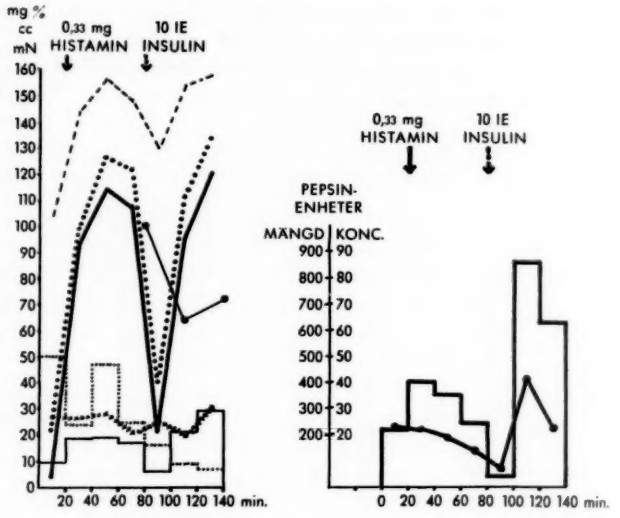


Fig. 10. Case No. 15. Age 8 9/12 years. Graphic presentation of histamine and insulin tests. For the sake of lucidity the diagram is put up in two parts representing the same child on the same occasion. (Signs same as in fig. 9.)

Case 21 was given avertin narcosis and in all the other cases chloral was used.

As an example of the graphic presentation of the test, case No. 27 has been chosen for this group (fig. 11).

Group IV. As an example of the graphic presentation of the test, case No. 32 has been chosen for this group (fig. 12).

Group V. All these cases got chloral narcosis. As an example of the graphic presentation of the test, case No. 41 (fig. 13) has been selected.

Comparative examinations with and without narcosis. In order to emphasize the importance of the child remaining quiet during the examination, the results obtained in case No. 20 with and without narcosis are given in fig. 14. The patient was troublesome during the test without narcosis. The test carried out some days later under avertin went off well.

Table 12. Group III. Children aged 4 $^{11}/_{12}$ to 1 year without any known disease of the stomach.

	No.	Sex	Age in years	Amt 60/min	Max amt/20 min	Max free HCl	Max Ta	Max chloride	Max PE/ml	Max PE/20 min	Amt PE/60 min
Histamine test	21	♂	4 $^{10}/_{12}$	35	17	114	127	148	20	190	340
	22	♀	3 $^{11}/_{12}$	58	34	115	126	160	22	260	640
	23	♀	3	22	12	131	139	156	25	190	340
	24	♀	2 $^{11}/_{12}$	19	8	127	137	151	14	90	190
	25	♀	2 $^{8}/_{12}$	34	15	133	143	156	50	270	530
	26	♂	2 $^{5}/_{12}$	29	18	133	143	162	62	240	590
	27	♂	2 $^{4}/_{12}$	20	10	121	131	154	29	120	240
	28	♀	2 $^{1}/_{12}$	32	13	97	111	147	9	110	260
	29	♀	1 $^{9}/_{12}$	19	9	104	126	150	41	120	290
	30	♀	1 $^{6}/_{12}$	12	6	122	134	151	16	70	150
	31	♀	1 $^{2}/_{12}$	11	5	106	118	143	9	40	80
Insulin test	21	♂	4 $^{10}/_{12}$		8	92	107	149	19	150	
	22	♀	3 $^{11}/_{12}$		15	129	140	160	26	340	
	23	♀	3		4	55	67	128	29	70	
	24	♀	2 $^{11}/_{12}$		14	35	49	103	8	50	
	25	♀	2 $^{8}/_{12}$		22	133	144	154	50	570	
	26	♂	2 $^{5}/_{12}$		5	130	140	156	20	100	
	27	♂	2 $^{4}/_{12}$		5	92	106	148	84	380	
	28	♀	2 $^{1}/_{12}$		5	86	101	142	19	80	
	29	♀	1 $^{9}/_{12}$		4.5	116	132	151	23	20	
	30	♀	1 $^{6}/_{12}$		5	89	106	144	29	120	
	31	♀	1 $^{2}/_{12}$		1	64	80	124	7	10	
Free HCl EWALD				12	26	10	11	18	1	16	2	0	7	7

Discussion.

Amount of gastric juice. Large variations were found in the amount of gastric juice within the same group of ages.

It is not impossible that the measured amounts of gastric juice are relatively too large for the examined children under 6 months of age. In all probability a greater admixture of saliva and mucus has taken place (dependent on technical difficulties) than for other children. The information regarding volume must therefore be taken with a certain amount of reservation and the somewhat lower HCl values in this age group also speak for eventual dilution for this age.

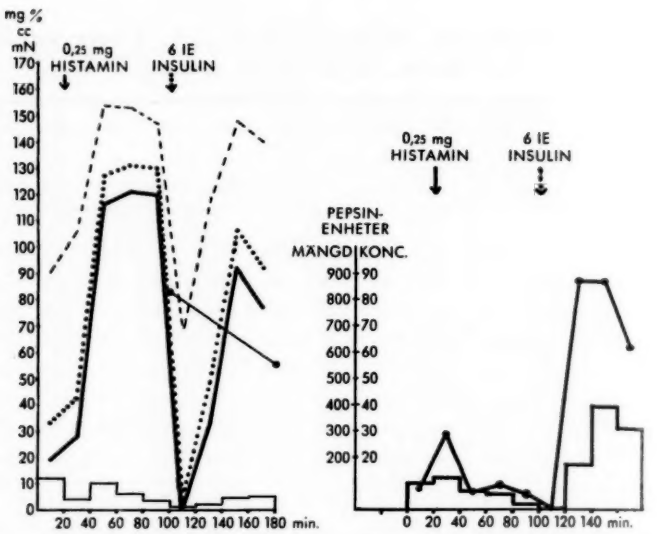


Fig. 11. Case No. 27. Age 2 4/12 years. Graphic presentation of histamine and insulin test. For the sake of lucidity the diagram has been put up in two parts which represent the same child and the same occasion of examination. (Signs same as fig. 9.)

Acidity. In fig. 15 below the highest values for free HCl have been marked with a · or a + irrespective of whether they have been obtained after histamine or insulin stimulation. The highest value was 139.2 mN (IHRE's highest value for adults was 147.8 mN). Successive media, connected by a broken line, are given for the different values. One notices that, to a large extent, this runs along the same level for the whole scale of ages.

In this examination the secondary acidity in children under 6 months of age has not quite the same high value as in other ages. This could indicate principally other secretory conditions in children under 6 months of age. FLENSBORG (1944) considers that the diffusion conditions in small children are different to those in older ones. This explanation could, in my opinion, be easier to accept if the changes in HCl values took place more gradually than is shown above.

Table 13. Group IV. Children aged between $^{11}/_{12}$ to $^{6}/_{12}$ years without any known disease of the stomach.

No.	32	33	34	35	36	37	38	39	40
Sex	♂	♀	♂	♂	♀	♀	♂	♂	♂
Age in years	$^{11}/_{12}$	$^{11}/_{12}$	$^{11}/_{12}$	$^{10}/_{12}$	$^{10}/_{12}$	$^{10}/_{12}$	$^{9}/_{12}$	$^{8}/_{12}$	$^{7}/_{12}$
Histamine test: Amt/60 min	25	26	18	8	12	10.5	9	16	4
Histamine test: Max amt/20 min	12.5	10	9.5	3	6	6.5	5.5	7	2
Histamine test: Max free HCl	130	136	99	127	95	111	81	101	107
Histamine test: Max Ta	139	146	111	138	109	125	94	119	118
Histamine test: Max chloride	155	160	146	154	137	152	133		143
Histamine test: Max PE/ml	14	25	14	16	6	9	37		37
Histamine test: Max PE/20 min	170	160	60	50	40	30	90		70
Histamine test: Amt PE/60 min	240	350	120	90	70	40	140		120
Insulin test: Max amt/20 min	3.5	2.5	5.5	1.2	5	3			1.2
Insulin test: Max free HCl	118	63	77	92	100	101			68
Insulin test: Max Ta	130	79	88	109	110	114			82
Insulin test: Max chloride	150	118	136	143	139	142			126
Insulin test: Max PE/ml	62	26	11	9	50	11			10
Insulin test: Max PE/20 min	120	60	50	10	250	30			10
Free HCl EWALD	21	5	10	1	1	7	0	0	3

In children under 6 months the primary HCl is probably secreted in such small quantities that secondary, acidity lowering factors could make themselves manifest in a higher degree than in other ages.

In general the total acidity in all children has been 10—15 units higher than free HCl. The highest value was 150.0 mN (IHRE had 152.8 mN for adults).

Chloride content. In the above graphic presentations of gastric juice (fig:s 9—14) one can see the good parallels between acidity and chloride content. In fig. 16 below (marked with a · or a +) the maximum chloride value of a gastric juice test taken from healthy children during a 20-minute

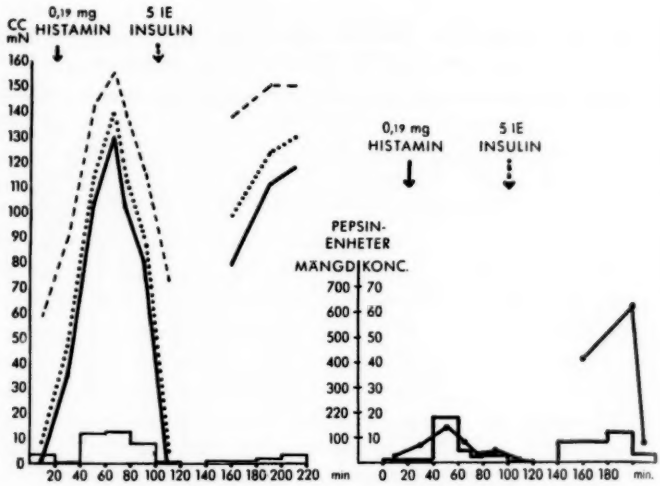


Fig. 12. Case No. 32. Age $^{11}/_{12}$ years. Graphic presentation of Histamine and insulin test. For the sake of lucidity the diagram has been put up in two parts which represent the same child and the same time of examination. (Signs same as in fig. 9.)

period are shown irrespective of whether they have occurred after histamine or insulin stimulation.

It appears that even the chloride values are generally alike for children in all ages. For children under six months they are somewhat lower, although the difference is less noticeable than with acidity. The highest value is 169.3 mN (the highest value for adults, according to IHRE, is 171.9 mN).

Pepsin content. Pepsin concentration per ml undiluted gastric juice, expressed in pepsin units (PE), have been marked in fig. 17 below. The values represent the highest concentration during a 20-minute period and are shown by a · for the histamine test and by a + for the insulin test.

The different values are levelled up by successive media which are connected by a line for histamine and by dashes for insulin values.

The size of the values, for the main part similar, are clear for all ages.

Table 14. Group V. Children from $^5/_{12}$ year to newly born without any known disease of the stomach.

No.			41	42	43	44	44[1]	45	45[2]
Sex			♂	♂	♀	♀	♀	♀	♀
Age in years			$^5/_{12}$	$^3/_{12}$	$^2/_{12}$	$^1/_{12}$	$^1/_{12}$	12 d.	12 d.
Histamine test	Amt/60 min		5	9	3.5	2	4	0	1.5
	Max amt/20 min		2.5	6	2	1	2	0	1.5
	Max free HCl		80	35	64	44	73		70
	Max Ta		92	48	76	57	83		81
	Max chloride		134	113	135	121	132		140
	Max PE/ml		10	7	7	7	5		6
	Max PE/20 min		20	30	10	5	10		5
	Amt PE/60 min		40	40	20	10	20		10
Insulin test	Max amt/20 min		2	5	1		1.5		0.5
	Max free HCl		74	41	37		65		54
	Max Ta		88	54	53		80		65
	Max chloride		128	116	136		134		135
	Max PE/ml		34	29	11		29		4
	Max PE/20 min		70	150	10		40		5
Free HCl EWALD			0	0	0				

[1] Increased doses of histamine and insulin (0.14 mg histamine per kg body weight. 4 IU insulin).

[2] Increased doses of histamine and insulin (0.20 mg histamine per kg body weight. 8 IU insulin).

The average of all maximum values for the histamine period is 18.7 ± 1.9 PE and for insulin 26.2 ± 2.8 PE. As is the case with the examinations of adults, an increase in pepsin values in children can be shown after insulin stimulation.

According to IHRE, 1 g crystalline pepsin corresponded to 34 600 of his units and by my method 1 g pepsin corresponded to 17 400 of my units. The figures for the concentration of pepsin and the amount of pepsin are, in my clinical investigations, on an average half as high as in those of IHRE. The

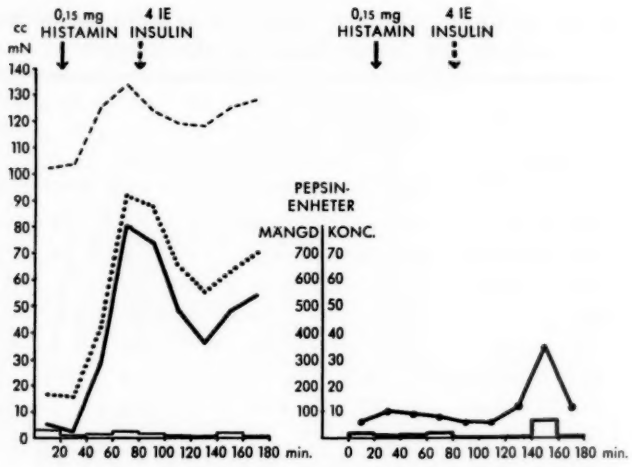


Fig. 13. Case No. 41. Age 5 months. Graphic presentation of histamine and insulin test. For the sake of lucidity the diagram is put up in two parts each representing the same child and the same time of examination. (Signs same as in fig. 9.)

absolute pepsin value in the gastric juice of children seems therefore to have the same order in size as in adults.

Synoptical table of results in histamine and insulin tests on healthy children.

Table 15 below gives the average within the respective age groups of the maximum concentration during a 20-minute period for some values. One can notice the good agreement in all age groups in the matter of HCl, chloride and pepsin. An exception is made for children under 6 months.

EWALD's test meal showed that the values for free HCl were low in small children and that they increased with rising age as will be seen from fig. 18 below, These values coincide well with normal values ordinarily given.

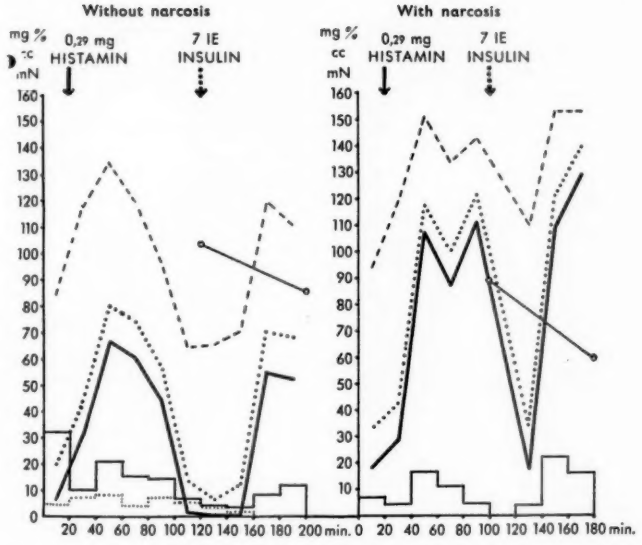


Fig. 14. Case No. 20. Age $6\,^{2}/_{12}$ years. Histamine and insulin tests were carried out on the same child with a two-day interval: once without narcosis and the other with narcosis. (Signs same as in fig. 9.)

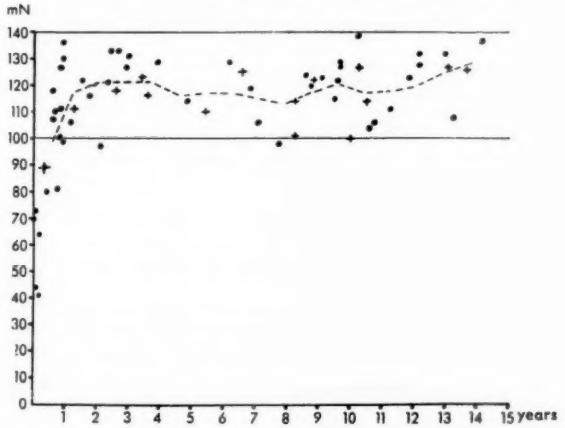


Fig. 15. Relation between child's age and maximum value of free HCl in a 20-minute test after histamine or insulin stimulation.
– – – – – = curve obtained by connecting successive media.

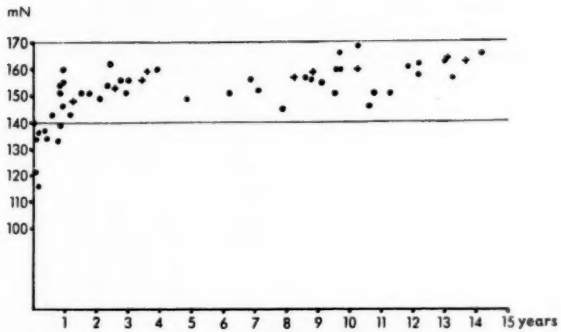


Fig. 16. Relation between age of child and maximum chloride content of gastric juice during a 20-minute period after histamine or insulin stimulation.

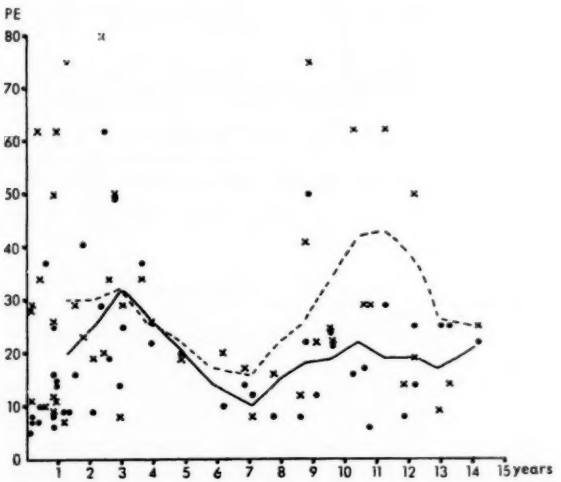


Fig. 17. Relation between child's age and maximum pepsin content, expressed as PE, in gastric juice in a 20-minute period after histamine (marked with a ●) and insuline (marked with an x) stimulation.

——— = connecting line for successive media of ●
– – – – – = connecting line for successive media of x

Table 15.

Group	Age, years	Histamine test		Pepsin units per ml	
		Free HCl mN	Chloride mN	Histamine test	Insulin test
I	14 $^{11}/_{12}$—10	119.3±4.4	156.1±2.9	18.7	31.0
II	9 $^{11}/_{12}$—5	116.1±3.1	155.4±1.7	15.2	20.8
III	4 $^{11}/_{12}$—1	118.4±3.7	152.6±1.7	27.0	28.5
IV	$^{11}/_{12}$—$^{6}/_{12}$	110.7±6.0	147.5±3.3	19.8	25.6
V	$^{5}/_{12}$—0	61.0±7.2	129.2±4.1	7.0	21.4

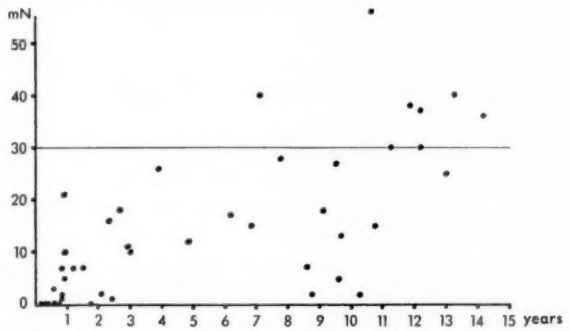


Fig. 18. Free HCl in EWALD's test meal in children of different ages (groups I—V). The majority of values lie between 0 and 30 mN which are marked by a fine, horisontal line.

Saliva amount.

The amount of saliva aspirated varied considerably but was often obviously large as will be seen from the figures below (tab. 16) which give the average amount of saliva aspirated during one hour.

It is obvious that a powerful decrease in the acidity, chloride and pepsin content of the gastric juice should have occurred if this saliva had been allowed to mix with the gastric juice. Saliva chloride remained at about 20 mN with a variation of between 11.4 and 37.1 mN.

Table 16.

Age (years)	Average amount of saliva (ml per hour)		
	10—14 $^{11}/_{12}$	5—9 $^{11}/_{12}$	1—4 $^{11}/_{12}$
Histamine period	51	40	7
Insulin period	29	19	7

Conclusions of the clinical investigations.

The results of these investigations appear to me worthy of the following conclusions.

1. Gastric juice examination with histamine and insulin tests and with perfect technique can be made on children of all ages.

2. The examinations on the younger children should be carried out under narcosis as a too great admixture with saliva and mucus can react unfavourably on the gastric juice of a restless child.

3. The secondary acidity in the gastric juice obtained after strong parenteral stimulation with histamine or insulin has an equally high, within certain limits, constant value for all ages.

4. No similarly high value for the secondary acidity has been obtained for children under six months as has been for other children.

5. The chloride content of gastric juice obtained after parenteral stimulation is equally high for all ages with the exception of children under 6 months who show a somewhat lower value.

6. The reason why the latter age-group has a lower secondary acidity and chloride content in the gastric juice than adults seems to be that the gastric juice is secreted in such small amounts that dilution, admixture with mucus and diffusion can act more strongly in the ventricle of small children than in adults.

7. On the whole, pepsin (the digestive power) is found in equally high concentration in the gastric juice of all ages. The pepsin concentration in the gastric juice after insulin stimulation is, in the majority of cases, higher than after histamine stimulation.

8. The examination thus entitles one to suppose that children in the complete scale of ages from birth to 15 years, have on the whole the same value for concentration of HCl, Cl and pepsin in the secreted gastric juice.

9. It will be seen from the previous conclusion that it is a mistake to put up, as a number of authors do, different normal values for concentration of HCl, Cl and pepsin for different ages in children. Any other normal value than that which reaches, or approaches a certain maximum does not exist. The possibility of obtaining such a normal value from a healthy child depends on the method of examination used.

10. In my material I have been able, by using a good method to arrive at a similar maximum value for all ages with the exception of children under 6 months of age.

PART III.

The Diagnostic Value of Gastric Juice Examinations in Children.

An orientated examination of gastric juice has been carried out on children with certain stomach troubles typical for childhood. In this respect special regard has been taken to the eventual advantage of a histamine or insulin test as well as for EWALD's test meal.

If one wishes to obtain an acceptable value with a histamine test it should be carried out in such a manner that sources of error, such as admixture with saliva etc., should be eliminated as far as possible. If this is not done the values will be far to varying and the diagnostic advantage lessened.

Previous examinations.

Gastric juice examinations on infants, even with different kinds of test meals, have hitherto been of very small diagnostic value.

In older children one has generally found a slight decrease in ventricle acidity in acute stomach trouble as well as a clear hypochlorhydria or achlorhydria in chronic stomach troubles.

Amongst the authors who have treated this subject one can find the following names in Scandinavian literature: AD. H. MEYER (1902), JACOBSEN (1920), CHIEWITZ (1922), KLEMENTSSON (1923), MUHL (1925), FORSSELL (1941) and FLENSBORG (1944).

There are only a few instances where the histamine test on children with stomach troubles are mentioned. FLENSBORG (1942, 1944) investigated a number of children suffering from gastrointestinal diseases with fractional histamine tests. He found, in almost every case, normal secretory conditions after histamine stimulation.

My own personal investigations.

In this connection, only a few preliminary results are quoted concerning a small number of cases showing, for childhood, characteristic symptoms from the digestive tract. The same methods of examination were used as for normal cases. The children were classified into two main types: A and B.

A. The following results (see tab. 17) were obtained from 7 children with abdominal pains of *the navel-colic type* and without objective symptoms from the ventricle. The explanatory signs in the table are the same as those used in normal cases previously described in Part II (see table 10).

Table 17.

No.			46	47	48	49	50	51	52
Sex			♀	♂	♀	♀	♂	♂	♀
Age, years			$13^{9}/_{12}$	$13^{1}/_{12}$	$10^{3}/_{12}$	$10^{1}/_{12}$	$8^{3}/_{12}$	$5^{5}/_{12}$	$3^{5}/_{12}$
Histamine test	Amount/60 min		87	63	44	78	53	52	26
	Max amount/20 min		43	28	15	24	27	29	12
	Max free HCl		126	127	127	100	101	110	123
	Max Total acidity		132	131	134	109	129	115	133
	Max chloride		163	164	160		157		156
Free HCl EWALD			30	42	17	3	0	8	3

In comparison with the normal cases, those with navel colic showed the same reaction to histamine.

B. In cases showing *hypochlorhydria or achlorhydria with* EWALD'S *test meal*, it was interesting to investigate to what extent a more powerful stimulant (histamine) could produce normal secretory conditions than ordinary food (test meal).

(a) Cases with *intestinal disturbances* of a slighter nature as, for instance, periodical diarrhoea, showed the following results (tab. 18).

Table 18.

No.	55	56	57	58	59
Sex	♀	♀	♂	♀	♂
Age in years	8 $^{8}/_{12}$	6 $^{7}/_{12}$	2 $^{7}/_{12}$	1 $^{3}/_{12}$	$^{4}/_{12}$
Histamine test: Amount/60 min	73	96	20	14	12.5
Histamine test: Max amount/20 min	26	35	9	7	6.5
Histamine test: Max free HCl	114	125	102	97	89
Histamine test: Max Ta	129	136	114	113	105
Histamine test: Max chloride			148	139	137
Histamine test: Max PE/ml			19	9	7
Histamine test: Max PE/20 min			140	70	30
Histamine test: Max PE/60 min			220	90	70
Insulin test: Max amount 20 min			9	6	1.5
Insulin test: Max free HCl			118	111	81
Insulin test: Max Ta			131	124	98
Insulin test: Max chloride			153	148	136
Insulin test: Max PE/ml			34	75	62
Insulin test: Max PE/20 min			130	220	60
Free HCl EWALD	0	0	0[1]	0	0

[1] Another day at 7, 12 & 17 o'c respectively 4, 4 och 6.

In these cases it was possible to show a hypochlorhydria or achlorhydria with EWALD's test meal but normal gastric juice value after parenteral stimulation (Histamine, insulin).

(b) The following values were obtained for a few cases with *slight or medium anorexia* (tab. 19).

It is not improbable that the appearance of achlorhydria in EWALD's test meal in these cases was the expression for a poor reaction in the ventricle from ordinary food. Achlorhydria was to such a degree apparent that a more powerful stimulant (histamine) was able to produce normal secretory conditions.

(c) The following values from histamine-insulin tests were obtained from two children with *severe anorexia* (tab. 20).

Table 19.

No.		60	61	62	63	64	65
Sex		♀	♀	♀	♀	♀	♀
Age in years		10 6/12	8 10/12	5 11/12	3 7/12	8/12	7/12
Histamine test	Amount/60 min	100	39	43	20	8.5	7
	Max amount/20 min	37	16	16	12	4.5	3
	Max free HCl	114	110	109	114	90	118
	Max Ta	123	128	119	127	99	128
	Max chloride		153	151	152	135	
	Max PE/ml		50		37		
	Max PE/20 min		800		450		
	Max PE/60 min		1250		480		
Insulin test	Max amount/20 min		14	9	14	3.5	
	Max free HCl		122	84	116	94	
	Max Ta		141	94	133	105	
	Max chloride		159	146	158	144	
	Max PE/ml		75		34		
	Max PE/20 min		600		210		
Free HCl EWALD		0[1]	0	0[2]	0	0	0

[1] At 17 o'c also 0. [2] Another day 7, 12, 18 o'c resp. 7, 7, 0.

In these cases there was obviously a hypochlorhydria not only after ordinary food but also after parenteral stimulation with histamine or insulin.

Variation in composition and time for test meal.

It is obvious that to get full advantage of EWALD's test meal, one must have regard to certain conditions which could influence the result of this investigation. In the first place I must point out the importance of adapting the size of the test meal to the age of the child.

It has been previously pointed out that the amount of gastric juice which is secreted in the ventricle after parenteral

Table 20.

No.		66	67
Sex		♂	♀
Age in years		2 2/12	2 1/12
Histamine test	Amount/60 min	8	7.5
	Max amount/20 min	5.5	5
	Max free HCl	16	6
	Max Ta	23	22
	Max chloride	124	119
	Max PE/ml	14	17
	Max PE/20 min	80	80
	Max PE/60 min	100	120
Insulin test	Max amount/20 min	4.5	5
	Max free HCl	35	0
	Max Ta	45	0
	Max chloride	124	129
	Max PE/ml	22	1.5
	Max PE/20 min	100	10
Free HCl EWALD		2	0.5

Table 21.

Weight of child kg	10	15	20	25	30	35	40	45	50
Rusks g	10	13	15	18	20	23	25	28	30
Water g	70	90	110	125	140	160	170	190	200

EWALD's test meal gradated in accordance with weight of child.

stimulation increases with rising age possibly in proportion to the surface of the stomach. The bread and water in the test meal has an acidity reducing action. It is therefore suitable that the size of the test meal varies in the same proportion to the amount of gastric juice in order that comparat-

ive values can be obtained. A suggestion for a gradated test meal after the weight of the child (and stomach surface) is given in table 21.

During recent years FORSGREN (1942—1943) has especially emphasized the importance of carrying out gastric juice examinations at different times during the day and not to pay attention to the value of one solitary examination in the morning. A person with achlorhydria in the morning can, for instance, have a full value at the midday test which can be expressed as a 24-hour rythmic function of the ventricle. For a smaller number of children the result is given here of examination with a test meal at different times of the day. In table 22 the value of free HCl only has been included, expressed in mN. (EWALD's test meal.)

Table 22.

Diagnosis	Age	8 o'c	12 o'c	18 o'c
Spasmophilia	$^{8}/_{12}$	2	1	8
Bronchitis asthmatica	$^{9}/_{12}$	0	0	3
Anorexia	$2\ ^{7}/_{12}$	4	4	6
Navel colic	$3\ ^{3}/_{12}$	2	0	3
Enuresis	$3\ ^{10}/_{12}$	40	2	10
Anorexia	$5\ ^{2}/_{12}$	9	0	0
Navel colic	$5\ ^{6}/_{12}$	9	0	2
Anorexia	$5\ ^{11}/_{12}$	7	7	0
Navel colic	$10\ ^{5}/_{12}$	15	16	5
Colitis spastica	$10\ ^{10}/_{12}$	10	0	0

Discussion.

Finally I will expound some points of view regarding the advantages and disadvantages of using EWALD's test meal and the histamine test in children both of which have been carried out in accordance with the methods mentioned above.

Histamine test.

Disadvantages. Is rather troublesome to carry out with reliable methods on children having regard to the fact that the saliva should preferably be aspirated and that younger children must be narcotized. It is a powerful stimulant which surely does not permit of small variations in functional disturbances in the ventricle coming to light. (If, however, one decreases the dose of histamine and relinquishes the demand to let the maximum value of acidity constitute a sign of a healthy gastric mucosa, one will obtain a lower and more varying acidity value. One can then just as well use a more physiological stimulant such as food.)

Advantages. In certain diseases with changes in the gastric mucosa such as is seen in gastritis as well as when investigating the state of the hypochylia, the histamine test is certainly of great diagnostic value. In the case of achlorhydria it is possible to ascertain if this is also refractory to histamine.

Ewald's test meal.

Disadvantages. The degree of admixture with mucus and saliva varies considerably and can give far too great a variation in the values for the stomach amount of gastric juice and its acidity.

Advantages. It is more simple to perform than one with a histamine test carried out by reliable methods. It is possibly more physiologically stimulant and can certainly allow small degrees in acidity and volume of gastric juice to appear than the histamine test.

Suggestion for a routine clinical examination of gastric juice in children.

1. Ewald's *test meal.* Should be given at different times of the day and in quantities of bread and water suitable to

the age of the child. If the value of free HCl obtained from this is approximately 10—40 mN (varying in accordance with the age of the children, see fig. 18) there are good grounds for the presence of normal function of the ventricle in the child.

2. If, on the other hand, an exceptionally high or low HCl value or achlorhydria is obtained, a *histamine test* is made.

When the value is low (with EWALD) there can be shown (with histamine):

(a) An achlorhydria refractory to histamine.

(b) A poor gastric juice secretion even after histamine. In this case it is probably a question of a lowered function of the ventricle.

(c) Normal gastric juice secretion after histamine despite hypochlorhydria with EWALD's test meal. Here lies possibly a certain lowering in the function of the ventricle in that a powerful stimulant alone is capable of giving normal gastric juice secretion.

When the value is high (with EWALD) there can be shown (with histamine):

(d) A rich gastric juice secretion after histamine often combined with a high acidity from EWALD's test meal. A similar result can be obtained from children with gastritis as well as from generally nervous, hypersensitive children in whose families there are quite often cases of gastritis or ulcus.

On the whole, when it concerns children, one should not be too hasty in making a diagnosis on the basis of one single gastric juice test as both EWALD's test and the histamine test can show great variations even normally.

Conclusions.

1. An orientated examination of children with symptoms from the digestive tract shows, in cases with so-called navel colic, fully normal secretory conditions after histamine stimulation.

2. The presence of hypochlorhydria or achlorhydria in subchronic intestinal disturbances and slighter anorexia seems to be apparent. After histamine stimulation in a limited number of cases examined by me, normal acidity values have been obtained. It is possible that the existing changes in the mucosa of children are, under these conditions, so small that a weaker and more physiological stimulant such as food is able, more easily than a powerful stimulant like histamine, to develop the degrees that, owing to the illness, exist in the manner in which the mucosa reacts.

3. In cases of severe gastritis one should be able to show a hypersecretion with an increased exchange diffusion in children by means of a histamine test.

4. Achlorhydria refractory to histamine appears, from my material, to be unusual.

5. EWALD's test meal should be given with an amount of bread and water adapted to the age of the child and the examination repeated at a couple of different times during the day. Acidity values in the stomach content varies not only from day to day but even at different times during the course of the same day.

6. When exceptionally high or low HCl value is evident or when there is achlorhydria from EWALD's test meal, the gastric juice examination should be augmented by a histamine test.

7. It can well be imagined that EWALD's test meal in children (given in accordarce with clause 5 above) can be a clinically enlightening test in cases (especially in those with a low HCl value with EWALD test) where histamine or other strong stimulants show normal HCl production.

References.

ANDERSEN, BENT: Undersøgelser over spæde børns ventrikelsekret. København, 1935. — CUTTER, R. D.: J. Ped. 1938, 12: 1. — DIETRICH, H. & SHELBY, D. C.: Am. J. Dis. Child. 1931, 41: 1086. — FLENSBORG, E. W.: Ventrikelsekretionen hos normale og syge Spæd- og Smaaborn. Roskilde, 1944. — IHRE, B.: Human gastric secretion. Acta Med. Scand. 1938. — KAIJSER, K.: Magsaftundersökningar på barn. Stockholm, 1943. — LINDERSTRØM-LANG, K.: Ref. Carlsberg Lab. 1931—33, 19: 1. — NEALE, A. V.: Arch. Dis. Childh. 1930, 5: 137. — SCOTT, G. H.: Am. J. Dis. Child. 1925, 30: 147. — TEORELL, T.: Untersuchungen über die Magensaftsekretion. Scand. Arch. Physiol. 1933, 66: 225. — ——: Act. Med. Scand. 1935, Vol. 85: 518. — ——: J. Gen. Physiol. 1939, 23: 263. — ——: J. Physiol. 1940, 97: 308.

From The Pediatric Clinic of Karolinska Institutet
at Kronprinsessan Lovisa's Children's Hospital.
Head: Professor A. Lichtenstein.

SOME TYPES OF NERVOUS DISORDERS IN CHILDREN AND THEIR PROGNOSIS

AN INVESTIGATION BASED ON THE STUDY
OF HOSPITAL MATERIAL, FOLLOWED UP
BY SUBSEQUENT EXAMINATION

BY

SIV GUNNARSON

ACTA PÆDIATRICA, VOL. XXXIII, SUPPLEMENTUM IV

Stockholm 1946

Received for print June 1945.

UPPSALA 1946
ALMQVIST & WIKSELLS BOKTRYCKERI AB
46339

Contents.

Page

Introduction 5

Literature 5

Different types of problem children according to different psychological schools 7

Prognosis and follow-up investigations of problem children 11

Own material 17

Behaviour problems 21

Organoneuroses 35

Summary of the therapeutic results 68

General summary 70

References 75

Introduction.

In children's hospitals a large number of children are being treated year by year for various nervous complaints, such as organic neuroses, behaviour problems, mental defects, etc. The hospital wards are being occupied in increasing degree by such children. This may be attributed partly to an actual increase of nervous disorders in children, due to various reasons, such as single-child families or broken homes, partly to the fact that parents and teachers, after much public enlightenment on the subject, now realize that many behaviour problems and school difficulties require expert medical treatment.

The results of the therapeutic measures taken by the hospitals, which may involve lengthy and expensive changes of environment, are of interest both from medical and social points of view. In order to contribute to the studies in this field, the material contained in the records of *Kronprinsessan Lovisa's Children's Hospital* has been worked up in regard to the children treated during a five years' period in that hospital. Despite the limitation of the material, we have considered these results to be worth publication, in view of the need of further information on this difficult subject.

Literature.

In the literature regarding nervous disorders in children we come across three diagnostic terms, namely psychopathy, neuropathy and neurosis. These terms are somewhat differently defined, and are used with different degrees of frequency, according as the authors have leanings towards pediatrics or psychiatry.

In the pediatric literature a distinction is made between

psychopathy and neuropathy and neurosis. »Psychopathy» is defined by GÖTT as a state caracterized by »constitutional psychical defects», whereas LICHTENSTEIN in »*Nordisk lärobok i Pediatrik*», applies the term to abnormal traits of character in the personality. »Neuropathy», according to BENJAMIN, designates functional anomalies, mainly of hereditary origin, in the nervous system, according to LICHTENSTEIN a state of abnormal nervous excitability, which colours the child's mode of reaction to the various irritants from the outer world, and which is already noticeable in the earliest stage of infancy before the child has as yet been exposed to any adverse exogenetic psychical influences. This hyper-excitability manifests itself especially in symptoms from the autonomic nervous system and is found in conjunction with so-called organoneurotic symptoms of various kinds (BENJAMIN, LICHTENSTEIN). Some organic neuroses, such as anorexia and vomitings, are regarded as mainly of environmental origin, whilst in other cases, for example in tic and disorders of sleep, they are considered largely to be due to hereditary factors.

In the psychiatric literature the term mostly employed is »psychopathy». According to SCHNEIDER, psychopaths are abnormal personalities, who suffer themselves from their defects, or whose abnormality is painful for those around around them. The term »neuropathy» is not employed in the psychiatric literature, and even »neurosis» is rejected by many (SCHNEIDER, etc.) as being ambiguous. HOMBURGER, in his psychiatric studies of children, like many other child-psychiaters, adopts SCHNEIDER's definition of psychopathy.

It should be observed, however, that also in adults we find numerous gradations between mental health and psychopathy. How much more difficult must it be to define psychopathy during childhood or to distinguish it from neuropathy, seeing that even under normal development children may come into conflict with their environment or pass through more or less lengthy crises. For such reasons, the term psychopathy, as well as its distinction from neuropathy, is tending to be discarded. SCHRÖDER, for example, rejects the term, pointing out that we are

not concerned here with sick children, but merely with »character variants» in children. These variants, he contends, can be understood and treated only from »characterological» points of view. SCHRÖDER therefore adopts the expression »charakterlich abartige Kinder» (cf LICHTENSTEIN's above definition of psychopathy). — BENJAMIN, rejecting the distinction between psychopathy and neuropathy, adopts the general designation »schwierige Kinder». In American literature the expression most frequently used in this connection is »problem children» (KANNER, POTTER and others). The term »neurosis» is retained, in which case it comprises organic states, or is replaced by »nervous symptoms from different organs».

Different types of problem children according to different psychological schools.

Problem children have been divided into different types according to many different principles.

The psycho-analytical school (FREUD and his successors), who refer all psychical aberrations in childhood to inhibited and suppressed impulses, desist from any classification of problem children into types. One of Freud's adherents, FRIEDLJUNG, however, sets up the following environmental types: the love-sated child, the love-hungry child, the contested child, the child rivalling with brothers and sisters, and finally combination types of these adverse environmental factors.

The individual psychological school (ADLER and successors) consider the lust of power to be the leading instinct in human beings and refer all behaviour problems to physical and mental defects in the child. Such defects give it a feeling of inferiority, which it tries to compensate by striving to become a central figure in the family. These psychologists set up two psychological types of child, the extroverted child, who acts first and then thinks, and the introvert, who thinks first and then acts (ADLER, JUNG).

It has been much discussed whether KRETSCHMER's theory of physical types and their mental parallels could be applied to children, in regard to whom constitutional investigations are complicated by the marked changes, physical and psychic, which the child passes through during the course of its development. Some authors consider that the difference between asthenic and pyknic habitus can already be detected during infancy (RUPILIUS), whilst others contend that it does not manifest itself until the play and school age has been reached (KRAMASCHE and KERCH, BENJAMIN). Many investigators, however, are unable to find any connection between physical and psychological types (SCHLESINGER, BENJAMIN, and others). KRASUSKY, on the other hand, found a good correspondence between physique and psychological types in children of all age-groups. NOBEL in »Innere Sekretion und Konstitution im Kindesalter», likewise states that Kretschmer's types, physically as well as characterologically, are already recognizable in childhood.

As regards the study of physical constitution in children, we may also mention the classification made by ZELLER, the pediatrician, and HETZER, the psychologist, which, however, refers merely to a short period in the child's life, namely the age of 6 to 7 years. ZELLER distinguishes at that age two physical types, namely the infant type and the school type. The infant type, he states, retains the softer and more rounded forms of the play age, with relatively short extremities and a large trunk, whereas the school type is characterized by more angular features and a lanker body, where the extremities are relatively longer and the trunk shorter.

HETZER, on the basis of similar material, studied the mental constitution of the child, and found that mental development proceeds parallel with the physical. Thus a child of the physical infant type has not attained the mental level of the school-child type, but gradually reaches that level when its body shows a transition to the last-mentioned type.

In other classifications into types, consideration is paid

only to traits of character or other psychological factors. Among them we note the following: —

Schneider classified adult psychopaths into different groups according to their salient traits of character, and Homburger, in his psychiatrical studies of children, adopts in essentials Schneider's typology. Thus he distinguishes nervous, timorous, weak-willed, unbalanced, callous and unemotional, irritable and disharmonious psychopaths.

Benjamin distinguishes three different types in children of all ages, namely the stubborn, the introvert and the regression type. In this classification he adopts, in essentials, the types set up by Charlotte Bühler for children during the first year of life. The *stubborn type* is characterized by tantrums, domineering, aggressiveness, tyranny, tormenting of animals and of other children, destructiveness, etc. Certain organo-neurotic symptoms, such as breath-holding spells, feeding difficulties and vomiting, are particularly frequent in the stubborn type.

The *introvert type* is characterized by shyness, reticence, lack of self-confidence and anguish. Children of this type avoid their school-fellows and try to escape from reality, they live in a world of phantasy and have a tendency to mendacity. In many cases they are liable to hypochondria and addicted to masturbation, nail-biting and thumb-sucking.

The main characteristic of the *regression type* is voluntary remaining at a previous level of development. Children of this type are dependent, unable to shift for themselves, inclined to frivolity and playing the fool, show lack of concentration and motor unrest. They often cause difficulties in passing from fluid to solid food, are liable to wetting and soiling till a comparatively late stage, and relatively often have enuresis and enkopresis.

Kanner cautions us against drawing up type classifications involving unduly narrow diagnostic limits, and stresses the importance of the principal symptom as the key to the diagnosis (»the complaint factor»). He thus bases the classification on the symptom because of which the child was sent to a doctor, and draws up a scheme of considerable range, with

the following main groups: I. Changes in personality, resulting from organic disease. II. Changes in personality, manifesting themselves in involuntary dysfunctions (headache, psychogenous vomiting, anorexia, obstipation, enkopresis, etc.). III. Changes in personality affecting the entire individual (intellectual disorders, disturbances in thought and feeling, disorders of speech, bad food habits, disorders of sleep, antisocial traits, sexual bad habits, spasms, hysteria, psychoses, etc.). — According to him, the advantages of this classification are that it is not based on any artificial limit between neuropathic and psychopathic, neurasthenic and psychasthenic states, and that its freedom from nosological restrictions makes it possible to take account of all the different combinations of symptoms. Thus diagnosis on the lines proposed by KANNER involves a reformulation of the symptomatology and should, he considers, give a clear presentation of the problem.

TRAMER classifies healthy children according to several different principles. Firstly he draws up a quantitative typology, in which he distinguishes different types according to the .tempo, energy and nature of the development. Secondly he distinguishes dynamic and static types. Finally, he sets up some characterological types: the motor type, the aggression type, the »attachment» type and the ego-type. Proceeding from this normal basis, he then distinguishes abnormal types within those groups. As regards psychopathic personalities in the years of youth, he adopts, broadly speaking, SCHNEIDER's typology (see above). He also mentions certain other types of importance in practice, such as enuretici, liars, thieves, prostitutes, etc.

TRAMER, in his classification of types during the age of childhood, refers repeatedly to SJÖBRING's views regarding mental disorders in adults and, though he does not himself adopt that investigator's system of classification, he considers that it might serve as a basis for at any rate a rough grouping of the mental disorders in children.

A classification on such lines has been made by LINDBERG, who groups mental disorders during the age of childhood ac-

cording to SJÖBRING's »minus variants». He thus distinguishes the following categories: the *sub-capable*, *i. e.* the intellectually defective, among whom he includes not only oligophrene and backward, but also physiologically dull children; the *sub-stable*, a group which practically corresponds to KRETSCHMER's cyclothymic type, and in which BENJAMIN's stubborn type may be included; the *sub-valid*, consisting of the asthenic personality type with asthenic states of insufficiency, and corresponding to BENJAMIN's introvert types; finally the *sub-solid*, who show hysterical personal traits and forms of insufficiency. It should be noted that BENJAMIN's regression group comprises mainly children with marked sub-solid symptoms. The abnormal motor types, as defined by TRAMER, should also be included in this category.

LINDBERG thus finds that many of the shifting states of insufficiency of functional nature during the age of childhood can be ranged within SJÖBRING's four »minus variants». He points out, however, that there are certain scarcely definable mixed states, where different »minus traits» partly cover one another and give rise to complex symptoms which cannot be explained on the basis of constitution psychology; and that there are several insufficiency pictures which, at our present stage of scientific knowledge, cannot be pressed into this, or any other known, system. He considers, however, that an analysis of personality on the lines proposed by SJÖBRING is of great value for reducing to order the diagnostics of child mentality, especially as the same diagnostic rules can then be followed both in children and adults.

Prognosis and follow-up investigations of problem children.

In regard to problem children, one must distinguish between socalled »situation prognosis» and prognosis of the child's future development (TRAMER). If, for example, one takes a child who is brought for examination because of difficulties

at school, the prognosis is primarily concerned with the prospects of settling the immediate school-conflict, that is to say, it is a »situation prognosis». Development prognosis, on the other hand, is, of course, greatly dependent on the time factor, being a judgment as to the probable effect of a certain passed mental disorder on the child's future development. Thus, to revert to the above example of difficulties at school, development prognosis judges how the child is likely to get on in the sequel, what kind of school is suitable, and how far it may be presumed that the child will grow up into a social or antisocial member of the community. In development prognosis many different factors come into play, primarily, of course, the child's future development, which may bring to light hitherto latent hereditary factors, environmental influences, therapeutic possibilities etc. (TRAMER).

As a basis for prognosis in regard to problem children of different types, carefully kept case-histories of each such child as well as enquiries, at frequent intervals, into the further destinies of the children are particularly recommended (KANNER, TRAMER and others). Child psychiatry, however, is a comparatively young science, and it is only within the last ten or fifteen years that »child guidance clinics» have sprung up at different places. Thus too short a time has elapsed for any extensive surveys of development prognosis. KANNER 1941, for example, whose clinic at Baltimore was opened in 1930, does not consider that the time is yet ripe for a thorough elaboration of his large material. He confines himself to certain casuistic reports, from which he does not venture to draw any prognostic conclusions.

The existing surveys of this nature are concerned mainly with delinquency in problem children and its prognosis.[1]

Among the few follow-up studies of problem children, we note the following:

FAIRBANKS at Baltimore in 1933 endeavoured to ascertain what had become of the children who 17 years previously had been examined for school problems of various kinds, such

[1] Cf. AHNSJÖ: »Delinquency in Girls and its Prognosis.» Act. pæd., vol. XXVIII, suppl. 3, 1941.

as backwardness, mendacity or petty pilfering. All of them had a low intelligence quotient, averaging 60 to 72 according to the Binet-Simon scale. The material comprised 166 children, of which 122 were traced. Their standard of life, capacity for work, delinquency etc. was compared with a normal group in which the intelligence quotient averaged over 90. It was found that the standard of life was surprisingly high among the backward, but that the group of normal intelligence held higher posts and showed less alcoholism, less public assistance, a smaller number of illegitimate or legitimate children, fewer divorces and less prostitution than the backward.

Gillespie and Lay in 1936 made a subsequent investigation of 80 children who two years previously had been treated at a child guidance clinic in London, and compared their improvement with that of a number of children in different schools who had shown similar behaviour problems, but had not been subjected to medical or psychological treatment. The investigation revealed that the children who had undergone such treatment had recovered in considerably larger numbers than the other group. Though the authors give no figures, the following data may be gathered from their report. The symptoms which had been most amenable to therapy were school problems in children of normal intelligence. Also disorders or defects in behaviour, such as refusal of food, truancy, petty pilfering, mendacity, vagrancy, destructiveness, etc., had in most cases been cured or ameliorated. Certain temperamental effects, such as shyness, hypersensitiveness or tantrums, had been easily mastered, whereas negativism and antisocial tendencies had been difficult to cope with. As regards disturbances of the autonomic system, disorders of sleep had readily responded to treatment, which had not been the case with enuresis. The authors had found that in certain cases enuresis could be cured by almost any medication, whereas in other cases all attempts at therapy had failed. Most cases, however, had recovered about puberty.

Howard W. Potter and Henriette Klein in 1937 made a follow-up examination of 175 children who had been treated

for various behaviour problems at a clinic in New York. All these children had been discharged from the clinic at least one year before the follow-up examination, which consisted in personal visits of parents and children to the doctor as well as visits of a social worker to the children's schools and homes. The tabular summary given by the authors indicates that out of the 97 patients who had been treated for behaviour problems without symptoms of encephalitis, cranial trauma, lues or other organic disease, 56.7 per cent showed a satisfactory social adjustment, whereas that of the others was unsatisfactory. The authors divided the children into three groups. The first group (25 children) comprised those who showed enuresis, masturbation, bad food habits, mild tantrums and non-aggressive disorders of behaviour; the second (51 children) comprised those who had behaved aggressively to persons or property, for example by destructiveness, theft, arson, or cruelty to other children or animals; also those who had been guilty of vagrancy or truancy; the third (21 children) comprised those suffering from psychoneuroses.

In these three groups, satisfactory social adjustment was shown by 56 %, 51 % and 71 %, respectively.

The authors mention that, when the material was divided in relation to social conclusions, the groups were too small to be judged. It seemed, however, that the prognosis was best for exceptionally gifted children (I. Q. 110 or more), for children in the prepuberal stage, for those who had shown a distinct improvement during the first six months after the commencement of the treatment, and finally where some fault on the part of the parents in the bringing-up of the child had been ascertained and could be rectified.

Nycander in 1938 made a follow-up examination of 101 children who had been treated 1—3 years previously at the Erica Foundation at Stockholm for behaviour problems. His material consists largely of children from responsible homes with a fairly good social standard. The author points out the large percentage of children in his material (75 %) who had grown up in an environment deviating from the normal, *i. e.*

that one of the parents was dead, that the parents had been divorced, that the children were illegitimate or adopted or from foster homes, that one of the parents was addicted to drink, etc. On subsequent examination 34 of the children were found to be free from symptoms, 50 improved, and 17 unimproved.

Schwalbe-Hansen in 1938 made a follow-up examination of 77 children who had been treated for psychopathy. The shortest period of observation was 4 years, the youngest patient at the examination was aged fourteen, and most of them were over sixteen. He found that 50 per cent of these children had developed favourably and that less than one-third of the total number still showed psychopathic traits. Most of those who had recovered had been brought up in a faulty environment.

Benjamin 1940, in examining 167 children who had been treated for stubbornness during the ages of 3—4 years, found that no less than 41.7 per cent of them came from single-child homes, and that 47.2 per cent of the others had merely one brother or sister. The majority of the children came from homes with a high standard of life and cultural level. On the follow-up examination of 42 of these children after an observation period of 6—8 years (in some cases only 4—6 years), he found in 83 per cent of them more or less marked behaviour problems, such as lack of discipline in school or home. Thus in all these cases the period of stubbornness had not been merely a transitory phenomenon, but had continued into a more advanced age. The prognosis was relatively favourable for the children that showed a high intelligence quotient.

Bender and Yarnell in 1941 published a report on the subsequent examination, after a five years' period, of 250 children who, during the pre-school age, had been treated at the psychiatric department of Bellevue Hospital. Half of these children were backward or otherwise mentally defective. The authors conclude from this examination that even marked backwardness may be found to be due to neglect and that

the intelligence quotient may rise when such children are placed in a suitable environment. They note further that in those children's homes where the children do not find affective contact, they are liable to mental disorders of an infantile, hyperkinetic and antisocial type. Such children subsequently adapt themselves badly in foster homes and schools, and constitute a serious menace to society, as a large percentage of them become antisocial. They also find that dull children adapt themselves worse in foster homes than the gifted; that neglected children are amenable to therapeutic treatment, their intelligence quota tending to rise under improved conditions; and that some gifted children from well-situated homes, where quarrels between the parents or immaturity in them lie at the back of the child's behaviour problems, are likewise ameable to therapy and develop well in the sequel.

Richards in 1941 published a report on a subsequent examination of 22 children who ten years previously had been examined for hypochondriac troubles at the Henry Phipps Psychiatric out-patient department, attached to the Johns Hopkins Hospital. At the time of the previous examination the children were between 3 and 15 years of age and the symptoms which they complained of had continued from 3 weeks to 9 years. They consisted of palpitation, gastric disorders, headache, pains in the chest or legs, weakness, giddiness, vomiting etc., with little or no objective basis. All of them, though devoid of psychopathic traits, were shy, oversensitive, suggestible and affect-labile. No anguish or the like could be observed in the children, but all of them had grown up in homes where the parents were suffering from divers physical complaints, so that the children were, so to speak, psychically infected. The therapeutic measures had consisted in the declaration that there was no objective basis for the subjective troubles, with the result that the attitude of the parents to the children's complaints was modified: improvement in school and home conditions: the inculcation of better habits in regard to sleep, meals etc.: and drawing attention to other joys in life, for children and parents, than indulgence

in complaints about sickness. In a few cases short changes of environment were required. The results of the follow-up examination after the lapse of ten years were that 19 out of 22 were completely free from symptoms, that two women were prostituted and one woman chronically hypochondriacal. These three women had been backward, and had not followed the therapeutic directions.

To compare the results of the above-mentioned follow-up examinations is scarcely possible, as the different authors have not adopted a uniform system of classification in regard to the children's nervous symptoms. In some respects, however, their findings seem to be fairly concordant. Thus the prognosis appears to be more favourable for a high than for a low level of intelligence, for organic neuroses than for mere behaviour problems. None of the investigators, however, have paid special regard to the way in which the prognosis may be affected by puberty, that is, the therapeutic results have been judged regardless of whether the children during the period of observation had passed the puberal stage or not.

Own material.

In 1936 there was a considerable increase in the number of children who were being treated for certain nervous diseases at Kronprinsessan Lovisa's Children's Hospital. Thus, whereas in 1935 the number of children treated in the pediatric wards, for intelligence defects, behaviour problems, organoneuroses and epilepsy was 9.4 per cent of the entire number, it had increased in 1936 to 16.8 per cent. During the following years this percentage has remained almost constant, these children averaging 15.5 per cent of the total.

The present investigation, which was started in the autumn of 1941, relates to children treated at that hospital during the years 1935 to 1939, and whose cases were diagnosed as neuropathy, psychopathy or enuresis. For the reasons stated below, children admitted from the year 1940 and the beginning of 1941 were also included among the enuretici.

The total number of children suffering from those complaints during that period was 411. Enquiries were made regarding the further destinies of these children, in order to ascertain how they had developed after discharge from the hospital. Contact was established with 337 children, 200 of whom personally presented themselves together with their relatives; enquiries regarding the others were made by visits to the homes, by telephone and by query papers sent to those residing in the provinces. As regards a number of children who had consulted the Stockholm Advisory Bureau for Educational Questions, valuable information was also obtained from its records. For this courtesy I am greatly indebted to the head of the Advisory Bureau, Dr. Torsten Ramer.

The purpose of these various enquiries was to ascertain how far it was possible to make a classification of the children into types; to investigate how certain groups of symptoms, such as enuresis, umbilical colic, etc., developed; what the prognosis would be for the sequel; what groups of symptoms could be distinguished among the problem children; whether the prognosis was more favourable for certain of these groups than for others; what symptoms seemed to be amenable to treatment, and in what cases the therapy had been without effect.

As regards problem children, a hospital material is in several respects one-sided and can by no means give a comprehensive picture. Thus, for example, organoneuroses will naturally form a very large percentage of the entire material, whereas children with school difficulties or other behaviour problems will in most cases be sent to Advisory Bureaus or out-patient departments. Moreover, a hospital material includes a large number of children from socially inferior homes, whence the influence of the bad environment will make itself felt in a far greater degree than as regards the patients of a private practitioner. It should also be noted that the children had been examined by many different doctors, and that in the case of mental disorders the judgment is bound to be more subjective than as regards organic diseases.

A hospital material will therefore be, in many respects, unequal and one-sided, but it also involves many advantages. Thus the children had been subjected to thorough physical examination, whence organic diseases could be excluded. Moreover, the observation period as a rule is rather long; during this time there are good opportunities for ascertaining home and school conditions, as well as for observation of the child in various natural situations, its behaviour during play and rest, alone and together with other children.

If one desires to form an idea of the future development of problem children, it is obviously desirable that as long a space of time as possible should have elapsed between treatment in hospital and follow-up examination. That for the purpose of investigation the five years' period 1935—1939 was selected was because about 1935 more detailed records were kept and a better system of registration was introduced, which has proved to be of considerable value in compiling the material. The longest period of observation in this investigation was 6 years, the shortest 2 years only. As its object was to ascertain the results of therapeutic measures, even the short observation period of 2 years was considered sufficient for that purpose. As regards future development, certain conclusions may also be drawn, as during the longest observation period of 5—6 years many of the children must have passed the puberal stage and taken up work for their livelihood.

The children who presented themselves personally were examined in accordance with the following method. A physical examination was made in each case and, where organic disease was suspected, was supplemented by urine and blood tests as well as by determination of the metabolism, electrocardiograms and roentgenography. The mental examination comprised firstly detailed anamnesis and »characterological» information supplied by a relative in the child's absence, secondly a talk with the child alone, in which case, when necessary, a short intelligence test, with stray questions according to the method of TERMAN, was made. In some ten doubtful cases the patient was sent to the wards for renewed

observation. All the children under twelve were occupied before the examination with coloured crayons and paper, being allowed to draw something of their own choice. This was found to be a good diversion while waiting; besides which, some of the drawings gave indications of interests, motorial dexterity or negativism, and in a few cases artistic talent.[1]

In compiling the material, the object was to make an unbiassed classification into types, uninfluenced by previous classifications. The children's symptoms and »characterological» data were noted on charts and, proceeding from the symptom for which the child had been sent to hospital, certain symptom groups were drawn up. Within these groups the »characterological» data were studied, in order to ascertain whether certain types of character show certain mental symptoms. In cases where the same child showed several nervous symptoms, for example, both anorexia and enuresis, each symptom was dealt with separately. The sum-total of the number of symptoms is thus larger than the total number of children.

The following main groups were distinguished.

	Number of Children.
I. Behaviour problems	175
II. Organic neuroses	
Anorexia	63
Umbilical colic	38
Vomiting	15
Heart disorders	5
Enuresis	109
Enkopresis	18
Tic	29
Headache	9
Disorders of sleep	40
Convulsions	11

As regards the results of the therapeutic measures, »recovered» denotes recovery in direct connection with those measures; »improved» signifies an improvement connected with the therapy, and which has lasted for at least

[1] No systematic study of the children's drawings was made.

one month; »unimproved» that no effect could be traced as the result of the therapy, even if the child had subsequently recovered.

Some other rules followed in compiling the material should be mentioned:

The intelligence tests made while the child was in the hospital were partly in accordance with Binet-Simon's method, partly in accordance with that of Terman. As the Binet-Simon tests usually give to high values for the intelligence quotients — as was shown also in this material in the thirty cases where both methods were employed for the same child —, an intelligence quotient under 90 according to Binet-Simon (under 80 according to Terman) was interpreted as backwardness, whilst a quotient below 80 according to Binet-Simon (under 70 according to Terman) was classed as imbecillity.

As regards hereditary factors, the classification into mental disease, psychopathy and »nervousness» on the basis of the anamnesis was made in accordance with the following rules. Mental disease was indicated where relatives had made a statement to that effect, or where the person in question had been admitted to a mental hospital. — It should be noted, however, that also persons who do not actually suffer from a mental disease, for example psychopaths, are treated at mental hospitals. — Alcoholism, sentence to imprisonment, confinement in residential homes or other grave cases of maladjustment to the community were regarded as indicative of psychopathy. Some backward people have probably been included in this group. Persons stated by a relative to be nervous or weak-nerved, but who had not been treated at a mental hospital, were termed »nervous». This classification is thus not free from error. The same remark applies to the control group of organically diseased children, with whom the nervous children were compared. This control group consisted of 100 children who had been treated at the hospital during the same period for organic diseases, such as pneumonia, kidney disease or angina, but who had noti shown any nervous symptoms during their stay there, or in the anamnesis.

Behaviour problems.

In regard to 175 children (109 boys and 66 girls) behaviour problems were one of the principal reasons for hospital treatment. The age distribution at the time of admission to the hospital is shown in Fig. 1, p. 26. All ages from infancy to puberty are represented, but most of them were in the preschool or school age.

The facts elicited by anamnesis and physical examination were substantially as follows: —

Hereditary disposition for mental disease, psychopathy and »nervousness» (Table 1) was observed in a considerably larger percentage in problem children than in those who had been treated at the hospital because of organic diseases.

Table 1. Heredity.

	Relative in mental hospital	Epilepsy	Psychopathy	»Nervousness»	Backwardness	Lues	Total
Problem children 167[1] . .	11.4 %	1.2 %	8.4 %	31.1 %	2.4 %	2.4 %	56.9 %
Organically diseased children 100	5 %	0	2 %	12 %	1 %	0	20 %

[1] As regards 8 no available information.

As regards the children's home conditions, they were in many cases unsatisfactory: the hospital records, however, contained detailed information only in a small number of cases. The number of broken homes, is remarkably large, as is shown by Table 2, which comprises children who had been adopted or in foster homes, whose parents had been divorced or were

Table 2.

	Children from children's home	Foster children	Adopted children	Divorce, parents dead, or mother unmarried	Other defects	Alcoholism etc.	
Problem children 175[1] . .	3= 1.7 %	12= 6.9 %	3= 1.7 %	38= 21.8 %	12= 6.9 %	15= 8.6 %	(7 cases doubly recorded) Total 47.6 %
Organically diseased children 100	0	4 %	0	15 %	0	1 %	Total 20 %

[1] As regards 5 no information available.

dead, or where the mother was unmarried. Figures are also given for the cases where alcoholism occurred in the family and, under the heading »other defects», some cases where the Child Welfare Board had reported neglect. These unsatisfactory home conditions were compared with similar shortcomings in the homes of 100 children who had been treated at the hospital for organic diseases. As shown by the Table, the number of cases where defective home conditions were recorded was more than twice as large among the problem children. (Cf. NYCANDER *supra*).

Eight of the children had been prematurely born infants, the others had been fully developed at birth. As regards development during the first years of life, 18 had shown a marked delay in learning to talk and 9 had not begun to walk till after the age of 18 months.

In the physical examination, symptoms of organic disease were found only in a few cases, where it had not been the origin of the behaviour problems; these problems had, in fact, existed long before, as in a few cases of mild broncho-pneumonia and mild heart failure, where those diseases had been the immediate occasion for admittance to the hospital. Among the cases suffering from vitium cordis, the heart affection was mild and in no case had led to invalid treatment. Only in a few cases of organic disease (asthma and adipositas) was there reason to presume that it had some connection with the child's behaviour problems. In three cases (1.7 %) the behaviour problems were certainly due to brain lesions, notably in two cases of congenital syphilis treated in infancy and in one case where the anamnesis indicated encephalitis.

In many cases there was marked dermographism and liability to perspiration, indicating lability in the autonomic nervous system. In the neurological examination exaggerated reflexes were observed in many cases.

Organoneurotic symptoms, such as anorexia, umbilical colic, enuresis, or the like, occurred in most of the children, in addition to behaviour problems. Only in 55 of them were they entirely absent. The frequency of the organoneuroses

Table 3. Frequency of organoneurotic symptoms in children presenting behaviour problems.

	Without organo-neurot. symptoms	With 1 org.-neur. symptom	With 2 org.-neur. symptoms	With 3 org.-neur. symptoms	With 4 org.-neur. symptoms
Number of children .	55	71	31	16	2
At follow-up examination:					
Easily educable . . .	30	45	20	9	
Difficultly educable .	24=43.6%	25=35.2%	11=35.5%	7=43.7%	2=100 %

in indicated in Table 3, which shows that one or two organoneurotic symptoms commonly occurred, but that some children had shown three or four such symptoms, concurrently with behaviour problems.

The Wassermann blood test was made in 76 cases with negative results; in one case of congenital syphilis the Müller-Ballung reaction was positive.

Roentgen examination of the skull was made in 23 cases. In one case a remarkably narrow skull-cap of peculiar form was observed, and in a case of adipositas the sella turcica was somewhat small. Otherwise the conditions were normal.

Intelligence tests were made in 114 cases, partly according to Binet-Simon partly according to Terman, in some cases according to both methods. Where such tests were not made, an approximate idea of the child's mental level was obtained from school reports (normal class or auxiliary class, school certificates, etc.) as well as by specimen tests according to Terman at the follow-up examination. 16 children were backward (9.1 %) and 8 imbecile (4.6 %), the others normally intelligent (86.3 %).

Behaviour problems in children, as we know, are due partly to constitutional factors, partly to environmental conditions, and in many cases are products of both these factors. In examining the children, endeavours were made to form an idea of the deeper-lying causes of the various nervous symptoms

shown by the child, and to ascertain how far the child's maladjustment had been mainly constitutional or mainly environmental, the therapy being adapted accordingly. The admission to the hospital had in itself been a therapeutic factor, in that the child had thus been removed from the conflict situation in which it had previously been involved. The duration of the stay in the hospital had varied from a few days to a few months, in most cases about a week or a fortnight. Other therapeutic measures are shown in Table 4.

Table 4. Therapy.

	Number of cases
Pedagogic advice	59
Improvement of home conditions	5
» » school »	16
Control at Advisory Bureau for Educational Questions	18
Convalescent treatment 1—2 months	25
Lengthy care at children's home ½—2 years	23
Private board for children, or children's home	15
Stay in the country with relatives	5
Foster home	9
Residental home	3
Asylum for imbeciles	3

»Improvement in home conditions» indicates that, in addition to pedagogic directions, some radical change had been made, *e. g.* that a new nurse had been engaged or a grandmother removed. »Improvement in school conditions» implies talks with teachers, change of teachers or school, or placing of the child in an auxiliary or observation class. Under the heading »Control at Advisory Bureau for Educational Questions» only those children who were sent there in direct connection with their stay at the hospital have been included. Many more children, before or after admission to hospital, had, on the initiative of the parents or school, visited the Bureau more or less frequently. In many cases treatment in the hospital had been followed by a stay at the convalescent home or children's home for a shorter or longer period.

A summary of the total length of the stay in hospital and any following convalescent treatment is given in Table 5.

Table 5. Survey of total length of hospital treatment and change of environment.

	0–1 w.	1–2 w.	2–3 w.	3–4 w.	1–2 m.	2–3 m.	3–6 m.	6–12 m	> 1 year
Number of children[1]	17	35	21	8	22	19	8	5	37

[1] As regards 3 no available information.

The results of the treatment are recorded in Fig. 1, which shows that the percentage of those who had completely recovered after treatment was largest in the youngest age-groups and had fallen with advancing age. The percentage of improved children is fairly similar in the different age-groups. Most of those who were unimproved after therapy were aged 3—4 years. No less than 36 per cent of the improved has relapsed.

In dividing the material into groups, different principles may be adopted. As pointed out in the review of the literature, the distinction between neuropathy and psychopathy is now being discarded, and greater caution is exercised in dia-

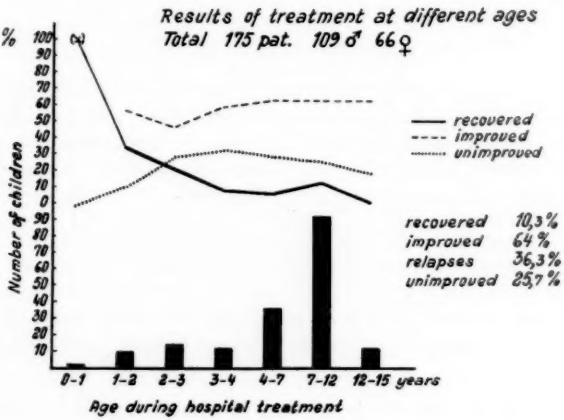


Fig. 1.

gnosing psychopathy in children. Nor could a division into mainly constitutionally defective children and mainly environmentally affected children be carried out in this material. The method adopted instead was to group the children according to their different reactions, as manifested in the various nervous symptoms (*Cf.* KANNER's classification, p. 9 above), and to observe what effect certain factors, such as hereditary disposition for nervous disease, certain home conditions, and the occurrence of organoneurosis and intelligence defects, have had on the results of the treatment.

Table 6. Frequency of some organoneurotic symptoms in different groups of children with behaviour problems.

	Stubborn screaming	Motorially restless	Sulky, cold-hearted	Delicate, tired, languid	Over-sensitive, anxious.
Anorexia . . .	15 %	18 %	12 %	4 %	15 %
Umbilical colic or vomiting . .	7 %	14 %	0	32 %	28 %
Enuresis . . .	25 %	41 %	38 %	10 %	15 %
Enkopresis . .	8 %	9 %	0	10 %	2 %
Tic	11 %	18 %	0	4 %	15 %
Disorders of sleep	15 %	5 %	0	23 %	17 %

Six different symptom groups were drawn up according to the dominant nervous symptoms shown by the children. The age-distribution in the different groups is shown in percentage in Fig. 2 p. 28. The largest group comprises the stubborn, screaming, fractious, quick-tempered children, who form 40 per cent of the total (cf. BENJAMIN's stubborn child, LINDBERG's substable children). Though all ages are represented, the number of children in the play age is strikingly large, which seems to be partly due to the greater stubbornness shown by children in that stage.

The next group comprises the children in whom motorial unrest had been particularly marked (cf. BENJAMIN's regression type, LINDBERG's substable children and TRAMER's abnormal

motorial type). These symptoms naturally made themselves particularly felt during school age, as it is in school that the children are required to sit still. To draw a line of demarcation between this group and the preceding group was in many cases difficult, as the motorially restless children often showed also stubbornness. They were, however, included in this group in cases where motorial unrest had been the dominant symptom.

Frequency of symptoms at different ages

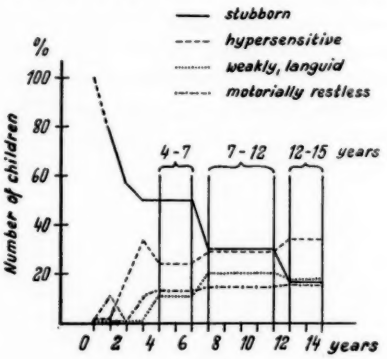


Fig. 2.

Delicate, languid, tired, peevish children form a group in which the majority are of school age (cf. BENJAMIN's introvert type, LINDBERG's sub-valid type).

One group consists of hypersensitive, nervous, anxious children, who are afraid of everything and everybody, who, for example, are terrified at having to go to the dentist, etc. (cf. BENJAMIN's introvert type and LINDBERG's sub-valid children). These states occur in all ages.

The last group comprises too small a number of children to serve as a basis for any curve of the age-frequency, and has therefore not been included in Fig. 2. It comprises 8 children who were sulky and cold-hearted and in whom callousness was the dominant symptom.

The number of children in the different symptom-groups and the sex distribution within those groups is shown in Fig. 3. Only in the group of delicate, languid, tired and peevish children are the girls in a majority, whilst in the other groups, as in the total material, the boys predominate.

The distribution of the organoneurotic symptoms into different groups is shown in Table 6 p. 27. The frequency of enuresis was most marked among the screaming, stubborn children,

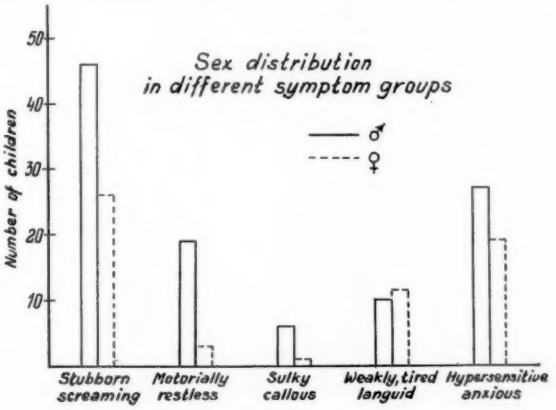


Fig. 3.

among the motorially restless and among the sulky, cold-hearted. Anorexia occurred in about the same degree in all the groups. Umbilical pains and disorders of sleep dominated in the delicate, tired and languid and in the oversensitive, anxious children.

The effects of the treatment are indicated in Fig. 4. The worst results were obtained in the sulky and callous group, where none of the children were free from their symptoms after the treatment and where the largest percentage of unimproved will be found. The next worst results are found in the group comprising the hypersensitive and anxious children. The largest percentage of improved is shown by the motorially restless and the stubborn children, who relapsed to approximately the same extent. (The percentage of relapses was reckoned

on the basis of the number of improved and correspondingly marked in the figure).

It is of great interest to see how problem children behave when they grow up, and how far they are able as adults to adjust themselves to the community. In this material none had past the age of twenty, though many had got beyond the puberal stage. The age-distribution at the time of the follow-up

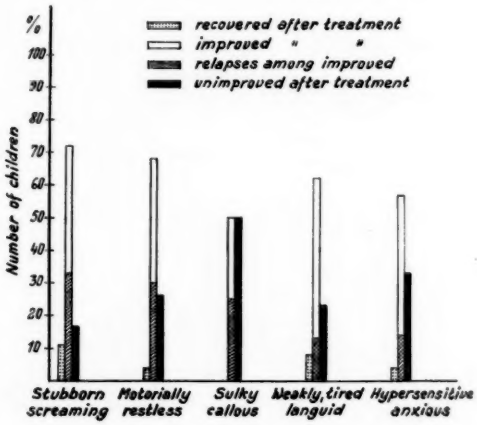


Fig. 4.

examination is shown in Fig. 5. At this examination enquiries were made from the parents as to how the children were now behaving, whether they were easily educable or not. The results of the enquiries from the parents will be found in Fig. 5 p. 31 where the number of easily educable and problem children at the time of the follow-up examination is shown in the different age-groups. Table 7 p. 32 shows what percentage of the children in the total material and in the different symptom groups were designated at that time as being easily educable or not. Somewhat more than half were easily educable, 40 % still showed behaviour problems, and in 1.2 % schizophrenia had developed. The largest percentage of the children who still presented behaviour problems will be found in the

cold-hearted and stubborn groups. It appears from Fig. 5 that the percentage of problem children is considerably larger in the prepuberal stage (49.6 %) than among those who had passed that stage (27.9 %). Table 8 p. 32 shows how many children at the time of the follow-up examination had been taken charge of by the local authorities.

In Table 9 p. 33 the symptoms at the time of the treatment

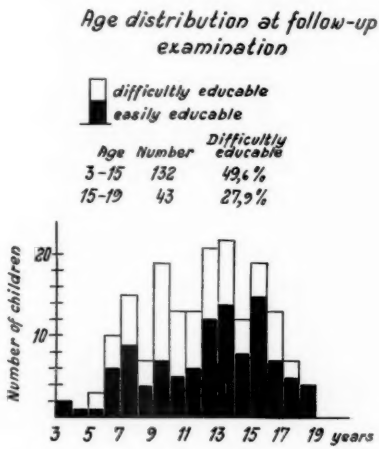


Fig. 5.

in hospital have been graded according to their severity and divided into slight, medium and severe. The table shows that out of the children who during treatment in the hospital had exhibited rather slight nervous symptoms, 15 per cent had continued to present behaviour problems, but that as many as 37 per cent of the worst problem children have become easily educable. This indicates that even grave behaviour problems may be due to maladjustment.

One may ask whether certain symptoms which in adults are usually designated as psychopathic phenomena, such as mendacity, petty pilfering, etc., tend to impair the results of the treatment also in children. In this material the number

Table 7. Number of children from the different symptom groups who, at the time of the follow-up examination were easily and difficultly educable respectively.

During hospital treatment	At follow-up exam.	
	easily educable	difficultly educable
Screaming, hot-tempered, rowdy, stubborn, 73 children	47.3 %	52.7 %
Motor. restless, 23 children	63.6 %	36.4 %
Sulky, callous, 8 »	12.5 %	87.5 %
Weakly, languid, tired, peevish, 23 children	77.3 %	22.7 % (incl. 1 schizophrenic)
Hypersensitive, restless, nervous, 48 children	74.5 %	25.5 % (incl. 1 schizophrenic and 1 dead

Total easily educable 58.5 % of entire material
» difficultly » 40.5 % » » »
Insane 1.2 %

Table 8. Number of children who at time of follow-up examination were in institutions.

	Number of children
Children's home	3
Home for problem children	1
Residential home	7
Asylum for imbeciles	3
Mental hospital	2
Arrested	1
	Total 17

= 9.7 % of entire material

of children with such symptoms, (see Table 10 p. 33) is too small to serve as a basis for any reliable conclusions. It seems, however, that these symptoms, particularly vagrancy, destruct-

Table 9.

During hospital treatment	At follow-up examination	
	Easily educable	Difficultly educable
Mild cases, 40 children . . .	34 = 85 %	6 = 15 %
Moderately severe cases, 69 children	46 = 67.2 %	22 = 31.4 % + 1 schiz. = 1.4 %
Severe cases, 66 children . .	25 = 37.5 %	39 = 59 % + 1 schiz. + 1 dead = 3.2 %

Table 10. Bearing of certain symptoms on prognosis for problem children.

	At follow-up exam. Easily educable	At follow-up exam. Difficultly educable
Mendacity	5	8 = 61.5 %
Petty pilfering	11	11 = 50 %
Vagabondage	1	4 = 80 %
Destructiveness	0	7 = 100 %
Incendiarism	1	3 = 75 %
Cruelty to animals	0	2 = 100 %
Entire material		40 %

iveness, a tendency to incendiarism, or cruelty to animals indicate the probability of an unfavourable course.

In Table 11, p. 34, the results of treatment of the hereditarily tainted children are compared with the results in children who had no such taint. The number of recovered, improved and unimproved children is approximately the same in the two groups, but the number of relapses is twice as large in the hereditarily tainted children as in the other group. A factor which appears to be of some importance in this respect besides the children's constitution is that many children with a hereditary disposition for nervous diseases, after the termination of the treatment, return to a nervous environment.

Table 11. Bearing of heredity on the prognosis.

Heredity for mental disease, epilepsy, psychopathy and »nervousness»		
Children without hereditary taint = 74	Recovered	10.8 %
	Improved	64.9 %
	Relapses	25 %
	Unimproved	24.3 %
Children with hereditary predisposition for nervous diseases = 93	Recovered	12.9 %
	Improved	61.2 %
	Relapses	47.3 %
	Unimproved	25.8 %
As regards 8 no available information		

Table 12. Heredity for mental disease, psychopathy or epilepsy.

	At follow-up examination	
	Easily educable	Difficultly educable
No hereditary taint, 74 children	66.2 %	33.8 %
Hereditary predisposition for mental disease, psychopathy or epilepsy, 41 children . . .	43.9 %	53.7 %

Table 12 shows the effect of heredity on the subsequent development of the children. Out of the children who had a hereditary disposition for nervous diseases (exclusive of »nervousness»), a considerably larger percentage were found at the follow-up examination to be less educable than those without that taint.

The results of the treatment of children from broken homes are no worse than the general average, nor is the frequency of relapses any larger. This may be attributed to the concentration of the treatment on the improvement of home conditions.

The prognosis of the organoneurotic symptoms was not

found to be less favourable in cases where they occurred in conjunction with behaviour problems (Table 3). Nor do the immediate results of the treatment of backward children diverge from the general average, for whom also the percentages of easily educable and problem children at the follow-up examination are in correspondence with the average for the total. In this material, however, the number of backward children is too small to permit any conclusions to be drawn.

Organoneuroses.

Nervous symptoms from the alimentary canal.

It has long been generally known that there is a close connection between mental phenomena and disturbances of the digestion. There is a very large literature showing how nervous disturbances may manifest themselves, wholly or partly, in symptoms from the alimentary canal, such as salivation, globus hystericus, aerophagy, rumination, anorexia, vomitings, abdominal pains, etc. When a nervous digestive disorder is suspected, it is important to be able to exclude an organic basis for the complaint. There has been a divergence of opinion as to where the line of demarcation between organic and nervous digestive disorders should be drawn. It was formerly considered that, in speaking of nervous gastric disorders, no secretorial or motorial disturbances should be found in the alimentary canal (Boas and others). Nowadays, however, we are not so strict in the delimitation of functional gastric disorders. True that the nature of the secretion of gastric juice in sick children has not yet been fully ascertained (cf. Kaiser). There is, however, reason to reckon with nervously induced disturbances of secretion in the alimentary canal, which may produce disease symptoms. It is considered, however, that disturbances of the secretion in certain cases may be of nervous origin, if there are no other signs of organic disease.

Also certain motorial disturbances in the alimentary canal

are considered to be of functional origin. Our knowledge that certain morbid symptoms in children are due to lack of autonomous nerve regulation has in fact led to the drawing-up of certain disease pictures, comprised under the name of spastic diatheses, which may be induced both by exogenous and endogenous irritations. Symptoms of spastic diatheses from the alimentary canal are recurrent umbilical colic, possibly with hyper-secretion and often also with spastic chronic obstipation. In such cases x-ray examination shows a spasm in the entire colon or a part thereof (LICHTENSTEIN). We are thus permitted to speak of nervous symptoms from the alimentary canal also in certain cases where disturbances of the secretion and spastic states in the stomach and colon are observed.

Anorexia and vomiting are regard by BENJAMIN as symptoms of stubbornness.

In the examined material the nervous symptoms from the alimentary canal were anorexia, umbilical colic and vomiting.

Own material.

Anorexia.

This group comprises the cases where lack of appetite had been one of the principal reasons for admission to the hospital and where no organic disease in the alimentary canal could be observed as the cause of the complaint. Disturbances of secretion and objective symptoms of a spastic nature were interpreted as functional phenomena. Cases where anorexia occurred as an independent symptom and where no precipitating cause could be shown, were termed »genuine anorexia». Cases where such a cause, *e. g.* an infection, could be observed, or where other symptoms as dominant as the anorexia, *e. g.* umbilical pains, occurred concurrently, were termed »secondary anorexia». 63 of the children subsequently examined had been hospitalized chiefly because of anorexia, which in 45 of them was of the »genuine» type. The distribution according to age

Table 13. Anorexia as a principal symptom (63 children).

	0—1 year	1—2 years	2—3 years	3—4 years	4—7 years	7—12 years	12—15 years	Total	
Age at time of hospital treatment	5	10	8	11	17	11	1	63=32 ♂ 31 ♀	
Recovered after discharge	0	3	2	2	4	4	0	15	23.8 %
Improved (14 d.) . . .	—	1	1	1	—	—	—	3	
» (1 m.) . . .	—	—	—	—	—	2	—	2	
» (more than 1 m.)	2	3	1	1	8	3	1	19	38.2 %
Unimproved	3	3	4	7	5	2	0	24	38.2 %

and sex is shown in Table 13. As will be seen, most of these children were below school age.

During the period when these children had been treated at Kronprinsessan Lovisas Hospital, the investigation, besides the ordinary routine, comprised the following procedure. Where the evacuation, on macroscopic inspection, was not normal, as well as in all the cases (30) in which Ewald's test-breakfast was administered, the feces were microscopically examined. Only in a few cases was an x-ray examination of the stomach and intestines or an abdominal survey considered necessary. The blood Wassermann was performed and intelligence tests were made in a few cases only.

The anamnesis and the physical examination gave substantially the following results. The weight at birth in all cases but three had been over 2 500 gr. Out of these three, only one had weighed less than 2 000 gr. In five cases the development during the first year of life appeared to be somewhat retarded, in one case it was much delayed, in the other cases normal. Many (40 children, 63.4 %) had received solely or mainly breast-milk, at any rate during the first five months of life, several of them for a longer period.

Anorexia usually began at an early age, especially as regards the »genuine» type, more than half the cases of which set in before the age of one year, often in connection with weaning or transition from milk to mash. Most of the others suffering from genuine anorexia had begun to refuse food at the age of two or three. In the cases of the secondary type, the anorexia usually set in somewhat later, merely in two cases

at the age of about one year. The immediate causes were infections or attacks of umbilical pain.

Children suffering from anorexia also showed other nervous symptoms. This usually occurred in conjunction with enuresis and disorders of sleep.

The physical examination in most cases did not reveal anything noteworthy. Most of the children were somewhat underweight in proportion to their height, but only in a few cases markedly emaciated.

In some of them systolic murmurs had been heard, but only in one case did the heart observations warrant the diagnosis of vitium cordis. In one case x-ray examination of the lungs revealed a primary complex; this case was nevertheless included in the »genuine» group, as anorexia had set in one and a half years before the commencement of the fever period, which was presumably caused by the lung affection; the anorexia moreover persisted for a couple of years after the child had been discharged from a sanatorium as recovered.

As regards the physical examination of the abdomen, nothing noteworthy was recorded. In the neurogical examination none of the children had shown anything pathological.

The blood investigation showed normal values, except in a case of secondary anorexia, where a slight anemia was observed.

Ewald's test-breakfast was administered to 30 of the children, in most of whom hypochylia or achylia was observed. As a fractional test-breakfast was administered in three cases only, I have thought it needless to enter into details on the subject.

In x-ray examinations a slight state of irritation in the stomach was observed in a few cases, but without any clinical basis for suspecting gastritis.

Most of the children appeared to be normally developed in regard to intelligence, so that testing was considered unnecessary. In the doubtful cases where such testing was made, it usually showed a normal intelligence quotient: only two or three children were of the auxiliary class standard.

As regards etiological factors in hereditity and environment, the following facts emerge:

Hereditary disposition for nervous diseases was found in a rather large percentage. In 7 cases there was mental disease in the family, in one case epilepsy, in 19 cases the father or mother or both were designated as nervous or weak-nerved (42.8 %). A hereditary disposition for gastric disease, on the other hand, was found merely in 3 cases.

As regards environmental factors, bad home conditions were found in 9 cases only. The composition of the family, on the other hand, seems to play a larger part in the origin of anorexia. One-third of the total number were single children (23) and more than half were first children (37). If we consider only the genuine anorexia, single and first children predominate still more. No less than 71 per cent of the children suffering from genuine anorexia were in fact first children, and out of them 50 per cent were single children.

The chief therapeutic factor in the treatment of children suffering from anorexia was the removal from the family to the hospital environment, where the child was no longer the centre of attention, as at home, but had been placed amongst other children, under the pedagogic influence of a trained hospital staff. Where hypochylia or achylia was observed, hydrochlorid acid was usually administered: otherwise, no medicine was given. In the severer cases, the change of environment was prolonged by a treatment at a convalescent home for some length of time. In some instances changes in the home, such as the engagement of a nurse, or change of the existing one, were recommended. As regards the smallest children, the mother or the nurse was allowed to come to the hospital and observe how the meals were arranged, and even to feed the children for a few days under supervision before the children were sent home.

At the follow-up examination, two-thirds of the children suffering from genuine anorexia were of school age or over it. The therapeutic results are shown in Table 13, p. 37, which includes all the children suffering from anorexia. After the treatment in hospital 23.8 per cent had completely recovered, whilst 38.2 per cent had improved. The unimproved cases amounted

Table 14. »Genuine anorexia». Therapeutic results after discharge from hospital.

	0—1 year	1—2 years	2—3 years	3—4 years	4—7 years	7—12 years	12—15 years	Total	%
Recovered after discharge	—	2	2	1	0	2	—	7	14.9
Improved » »	2	4	2	2	2	5	1	18	38.3
Unimproved » »	3	3	2	7	5	2	—	22	46,8
Relapses » »	1	1	1	2	0	1	—	6	33

to 38.2 per cent. If we consider only the cases of genuine anorexia, the results are worse (see Table 14). Only 14.9 per cent of them had recovered after hospital treatment, 38.3 per cent (of whom 33 % had relapsed) had improved, and 46.8 per cent had remained unimproved.

In a survey of all the cases, no difference in the therapeutic results of a shorter or longer change of environment can be observed. If, on the other hand, we confine ourselves to the genuine anorexia (Table 15), the longer change in environment appears to have had a 10 per cent better effect. Seeing that in practice it is only the most serious cases for which a lengthy change is recommended, somewhat better re-

Table 15. »Genuine Anorexia». Therapeutic results relatively to duration of change of environment.

Duration of change	0—1 w.	1—2 w.	2—3 w.	3—4 w.	1—2 m.	2—3 m.	3 m.—½ year
Recovered after change . . .	1	1	—	2	3	—	—
Improved » » . . .	—	5	3	1	4	4	3
Unimproved » » . . .	2	1	4	4	7	1	2
Relapses » » . . .	—	3	1	—	1	2	—

Percentage results: Change of env. 1—3 w. Rec. and impr. 35 %
3 w.—½ year rec.+impr. 45 %

sults from the longer changes of environment seem to be indicated.

No difference in therapeutic effect between the cases where the anorexia set in during the first year of life and those in which it appeared later has been observed.

In 33 cases the parents, in reply to enquiries, were able to state an exact date when the anorexia had ceased. 32 out of these 33 children had recovered before, or at, the age of seven. The usual answer to this question was that the anorexia had ceased in connection with school-feeding.

No special type of child affected with anorexia could be observed throughout. A strikingly large number of children suffering from anorexia, however, were characterized as lively, screaming, independent, stubborn and affect-labile. They are liable to tantrums if anything goes against them, and they are apt to tyrannize their surroundings. They correspond, broadly speaking, with BENJAMIN's stubborn types or LINDBERG's sub-stable type. Some of them are also rather reserved, do not get on well with other children, and therefore prefer to go about alone or have merely a few playmates.

Half of the total number of children with anorexia continued to show behaviour problems in the sequel, and at the time of the follow-up examination were designated by their relatives as problem children. Among the more serious behaviuor problems we note destructiveness, mendacity, petty pilfering, cowardice and jealousy. The relative proportion between easily educable and problem children is the same among those who had attained school age and those who were still in the play age.

As above mentioned, many of those suffering from anorexia were first or single children. The further development of these children, however, was not unfavourable as compared with that of children from larger families: in both groups half of them presented behaviour problems.

Genuine anorexia was not found to have any special correlation with other organoneuroses. Only eight of these children had enuresis.

Secondary anorexia, as above indicated, often occured in conjunction with umbilical colic.

Umbilical colic.

This group comprises the children who, on admission to the hospital, had been suffering from pains in the umbilical region and where no organic cause could be shown. In addition to the colic, vomiting occurred in a few cases, but in all of them the pains were the dominant symptom.

Umbilical colic occurred in 38 children (14 boys and 24 girls). The age-distribution is shown in Table 16, which indicates that most of the children were of school age.

Table 16. Umbilical colic.

38 children (14 boys and 24 girls.)

Age group	0—1 year	1—2 years	2—3 years	3—4 years	4—7 years	7—12 years	12—15 years	Total	Percentage
Onset at age	0	0	1	1	8	27	1	38	—
After discharge:									
Recovered	—	—	—	1	4	14	1	20	52.7 %
Improved	—	—	1	—	3	10	—	14	36.8 %
Relapses	—	—	1	1	0	11	—	13	38.2 %
Unimproved	—	—	—	—	1	3	—	4	10.5 %

As regards the routine examination, the reader is referred to the preceding section on anorexia. All of them were subjected to the tuberculin test. Ewald's test-meal was administered to 30 of the children, X-ray examination of the stomach and intestines was performed in ten cases, and an abdominal survey in the same number.

In most cases nothing positive was revealed in the physical examination. Several of the children were underweight in proportion to their height, but only two or three markedly emaciated. In several cases systolic murmurs were heard, but only in two of them was mild congenital heart disease observed. Merely a few of the children had shown tenderness on palpation in the umbilical region. In the neurological examination exaggerated reflexes were observed in about 10 per cent of the cases; one of the children showed chorea-like movements, though actual chorea could be ruled out.

Another child showed nystagmus. The tuberculin test in most cases was negative, only three of the children being tuberculin-positive. In one of these children calcified glands in the abdomen were observed by the x-ray-examination, but no signs of an active tuberculous process in the abdomen, and in another the abdominal survey was negative. In the third case no abdominal survey was made, as the child showed no signs of organic abdominal affection.

Out of the 30 test-breakfasts administered, 28 were according to Ewald's method and only two were fractional. 50 per cent of the children showed hypochylia, none of them were completely achylous. The feces examination did not reveal anything pathological.

The x-ray examinations, which were made only in a small number of cases, showed in two of them slight spastic states in the stomach and colon, without any clinical basis for suspecting gastritis or colitis. The Wassermann test was performed in 9 cases, all of which were negative.

Intelligence tests were made in 13 cases: out of these, two children were backward and one imbecile. The other children appeared to be intellectually normal.

In 11 cases (26.3 %) nervousness or mental disease occurred in the family. In 4 cases one of the parents had an intestinal or abdominal disease. Bad home conditions were not particularly marked in this group; on the other hand, the effect of the school environment was striking: in no less than half the cases school difficulties had been the precipitating cause of the abdominal symptoms.

As regards the treatment, see the previous section on anorexia. The principal therapeutic factor was a change of environment and the information that the disturbing abdominal symptoms gave no real cause for alarm. In some cases hydrochlorid acid was prescribed for children suffering from hypochylia. The change of environment was sometimes prolonged by treatment at a convalescent home. Phenemal and eumydrin pills were prescribed for six of the children after discharge from the hospital.

The results of the treatment are shown in Table 16, p. 42, Half of the children recovered immediately after treatment in the hospital and merely 10 per cent had remained unimproved. It should be mentioned, however, that a large percentage of the improved had relapsed. Changes of environment for a

considerable length of time had no better effect than shorter changes. Out of the six children for whom phenemal and eumydrin had been prescribed, four had recovered almost immediately and two had improved. The number of these cases, however, is too small to permit any conclusions.

A survey of the characteristic data shows that the children suffering from abdominal pains were usually lively, nervous, sensitive and affect-labile, rather delicate in constitution. In half the number of cases the abdominal symptoms, as already stated, had been precipitated by school difficulties. Most of the children were designated by the parents at the time of the follow-up examination as easily educable (79 %), though many of them were still rather sensitive and nervous. Psychopathic traits were observed merely in some few cases.

Vomiting.

This symptom group includes the children sent to the hospital for investigation of the cause of vomiting and in regard to whom an organic origin of the vomiting could be excluded.

Vomiting as a principal symptom occurred in 15 children, some of whom were infants, the others of school age (Table 17). One-third of them were boys and two-thirds girls.

Table 17. Vomiting.

(5 boys, 10 girls).

Age group	0—1 year	1—2 years	2—3 years	3—4 years	4—7 years	7—12 years	12—15 years	Total	%
Age at first attack	4	0	1	1	1	8	0	15	
After discharge:									
Recovered	1	—	1	1	—	3	—	6	40 %
Improved	3	—	—	—	1	3	—	7	46.6 %
Relapses	—	—	—	—	—	1	—	1	14.3 %
Unimproved	—	—	—	—	—	2	—	2	13.4 %

A regards the routine examinations, see the section on anorexia.

In most cases the physical examination did not bring to light anything particular. Half the children were underweight in proportion to their height, but merely two or three markedly emaciated. In a few cases systolic murmurs were heard, and in one of them were interpreted as indicative of a mild congenital vitium cordis. None of them showed any objective abdominal symptoms. A few children had exaggerated reflexes. One of the children had been treated in infancy for congenital syphilis, but was negative to the Wassermann test long before admission to the hospital. All the children were tuberculin-tested with a negative result. Ewald's test-breakfast was administered to 10 children, all of whom had free hydrochlorid acid with values within the normal limits of variation. X-ray examination of the stomach and intestines was performed in two cases. In one of them a slight state of irritation was observed in the stomach, but without any chemical basis for suspicion of gastritis.

Intelligence tests were made in 7 cases. One of the children was of the auxiliary class standard, whilst the other tested children, as in all probability those who were not tested, were of normal intelligence.

In 40 % nervousness or mental disease occurred in the child's family. None of the parents had had any gastric disease.

The cases are too few to permit any study of the different age-groups. In the anamnesis, a marked nervous unrest was noted throughout: as regards the infants, it had manifested itself in screams, anorexia and restless sleep; in the older children the symptoms were anorexia, fatigue, nervousness and general oversensitiveness. The vomiting was considered to have been caused, in all the school children, by school difficulties. Most of those children had in fact felt sick and vomited before going to school.

The treatment naturally varied in the different age-groups. The infants and children in the play-age were subjected to a good regime, and in a few cases a change of environment for some length of time was prescribed. As for the school children, attempts were made in the first place to improve the school situation and in some cases small doses of phenemal were administered.

The results of the treatment are given in Table 17, p. 44, which shows that 40 per cent of the children had recovered

direct after hospital treatment, 47 per cent had improved and 13 per cent had remained unimproved. The material is too small to warrant further elaboration.

In surveying the characteristic data, it will be found that the children are of the same type as those suffering from umbilical colic, *i. e.* are lively, nervous, sensitive and affect-labile, rather delicate in constitution. At the follow-up examination most of them (86.6 %) were found to be easily educable, though many were still sensitive and nervous.

Nervous symptoms from the heart.

Five of the children, all of whom were girls, showed nervous symptoms from the heart. The age distribution is shown in Table 18.

Table 18. Nervous symptoms from the heart.
(5 girls).

	4—7 years	7—12 years	12—15 years
Number	1	3	1
Recovered	1	2	—
Improved	—	—	—
Relapses	—	—	—
Unimproved	—	1	1

In all these cases organic heart disease could be excluded after physical examination, electrocardiography and x-ray examination of the heart. All five were of normal intelligence.

The treatment consisted solely in the imparting of information regarding the functional nature of the complaints, and a change of environment.

The results are given in the table.

At the time of the follow-up examination, the three who had recovered in direct connection with the hospital treatment were found to be easily educable. As regards the two who had remained unimproved, schizophrenia had gradually developed

in one of them, the other was a girl with various severe behaviour problems.

The cases are, of course, too few for any character analysis.

Enuresis.

There has been much discussion as to whether enuresis is to be regarded as a symptom of disease of mainly organic nature or as functional complaint.

Among the organic causes which have been indicated, we note firstly congenital malformations, such as phimosis, unduly short frenulum, spina bifida and myelodysplasia, secondly diseases in the urinary system such as balanitis, vulvitis, cystitis, vesical calculus, tuberculosis, oxyuriasis, etc. Finally, enuresis is considered by some French authors to be, in many cases, a symptom of syphilis. PINARD, for example, among 100 enuretici had found syphilis in 77 per cent; also German investigators, trying to find an organic cause, have latterly adhered to this view. Thus GRAFE, among 85 cases of enuresis found symptoms of syphilis congenita in 74 per cent, whilst KISLICENKO, among 26 such cases, suspected congenital syphilis in no less than 92 per cent.

Other authors, opposing these attempts to find an organic cause of enuresis, point out that in most cases it is simply a nervous symptom. Thus KARGER considers that phimosis and pseudophimosis have been considerably exaggerated as etiological factors, and that only in cases where eczema has been observed on glans penis, can they be supposed to be causal factors. KARGER considers that myelodysplasia has no connection with enuresis, but stresses the importance of looking for pyuria, tuberculosis and colds as causal factors. HINZE contends that spina bifida plays no part in the origin of enuresis and, on the basis of a large material, maintains that it is found quite as often in nonenuretici. According to LICHTENSTEIN, the part played by spina bifida has not yet been definitely ascertained.

Among psychogenous causes of enuresis, we note mental

backwardness, psychopathia and neuropathia. According to BENJAMIN, enuresis is due to faults of education, although he presumes that certain children such as those who are backward or mentally abnormal present special difficulties in bringing-up to cleanliness. He regards enuresis as a regression symptom, the child reverting to, or remaining at, an earlier stage of development. He finds that enuretici are of a certain type, notably the lax, weak-willed, careless and inactive children. TRAMER, on the other hand, contends that this complaint is not confined to any special type, and that enuresis may occur as a nervous symptom in children of any type of character.

Experience in belligerent countries among evacuated children supports the view that enuresis in many cases is amenable to education, and that emotional factors also play an important part. Thus, enuresis sometimes occurs in evacuated children who had previously been dry, because they lose their hold on life when they are removed from their homes and transferred to a foreign environment (ODLUM). According to English experience, enuresis has been found to be more common among children from socially inferior homes. Finally, the origin of enuresis is stated to be due to »mental infection» (GRUNDTOFT).

The depth of sleep has also been much discussed, and some authors consider that children affected with enuresis sleep very deeply and are difficult to awake (KARGER, BENJAMIN).

It has also been pointed out that mentally normal children may show enuresis, and that in many cases it may be a question of lack of nervous control of the bladder, a supposition which has latterly been supported by intra-vesical pressure measurements (LICHTENSTEIN).

Enuresis often occurs in conjunction with enkopresis, especially in imbecile children and idiots, sometimes also in children of normal intelligence (concerning enkopresis see the following chapter).

As regards the therapy, many different methods have been tried, with greatly varying results. Some investigators have

obtained excellent results with the most varying methods of treatment, whereas others view their therapy with greater pessimism. LICHTENSTEIN draws attention to two distinct types of enuresis, differing in respect to prognosis, namely children who have learnt in due time to keep dry, but who after physical or mental strain have relapses of varying duration, and secondly those who have never learnt to control the voiding of the bladder. According to him, the last-mentioned children, from a therapeutic point of view, present considerably greater difficulties than the first-mentioned.

Own material.

The age at which a child learns to control the bladder day and night varies considerably, and one may question where one should draw the line of demarcation between the inability of the child during the first years of life to control the voiding of the bladder, and the symptom of enuresis. Some authors put the age at which one can expect that children should be dry as high as four years (CAMPBELL), but as a rule the line is drawn at the age of three (HOLT, LICHTENSTEIN, TRAMER). In this material the children who have not been dry night and day after the age of three, have been termed enuretici.

Table 19. Enuresis (all cases). Therapeutic results.

Age at time of hospital treatment	2–3 years	3–4 y.	4–7 y.	7–12 y.	12–15 y.	Total	Percentage
Number	8	13	37	44	7	109	—
Recovered	—	2	4	2	—	8	7·3 %
Improved							
for a fortnight	—	—	2	5	—	7	38.6 %
» 1 month	—	—	2	1	—	3	
» longer period . . .	—	4	12	12	4	32	
Unimproved	8	7	17	24	3	59	54.1 %
Relapses	—	1	7	10	1	19	45.5 %

109 children treated at the hospital in the course of the period from 1935 to the beginning of 1941 were subjected to a follow-up examination. The shortest period of observation was merely six months, but as all the relapses had occurred earlier than six months after discharge from the hospital, such a brief period is considered to be sufficient.

The age-distribution at the time of the hospital treatment is shown in Table 19, p. 49.

The children who were treated at the hospital for enuresis before the age of three were included in the material only in cases where they had continued to show enuresis after that age. The procedure was substantially as follows. A special examination of the external genitalia was made, and in all cases the urine was examined for sugar, albumin or sediment. In 60 cases the lumbar vertebrae and sacrum were x-rayed, in 46 cases intravenous urography was performed and in 48 cases intelligence tests were made.

The anamnesis and status show the following: Half the number of children had a nervous hereditary taint and in 22 per cent of them (24 children) enuresis had been observed in the family. As regards one-third of the children the home conditions had been remarkably bad. With one exception, all the children had been fully developed at birth, and the delivery had been normal. The development during the first year of life had been retarded in 15 cases, but merely in one case greatly delayed. Most of the children (75) had been sent to the hospital because of enuresis, others (22) for various behaviour problems. 20 children, in addition to enuresis, had also shown incontinentia alvi. In some cases the children had been hospitalized because of acute infection or the like, and the occurrence of enuresis had then been recorded as an accessory symptom. The children who had been admitted to the hospital because of enuresis, with or without other neuropathic symptoms, had usually shown normal conditions on physical examination. Five of the children had symptoms of heart disease, one of them showed a hypopituary dwarfishness, and another a spastic paresis in one arm, of unknown origin. Malformations or morbid processes in the external genitalia

were found merely in a few cases: two of the children showed hypospadia, one of them a unilateral cryptorchidism and three phimosis. One case of enuresis after poliomyelitis had been excluded; three children had oxyuriasis.

In the smaller group which had been hospitalized because of infections, symptoms of those infections had been found in the physical examination, otherwise nothing special. In all these cases enuresis had occurred previous to the infection and thus cannot have been caused by it.

The urine finds were negative with the exception of two cases where a transitory glycosuria was observed, though with normal glucose tolerance tests and blood-sugar values, and two cases where bacteriuria had been found.

The blood Wassermann was performed in 31 cases, with negative results. No congenital syphilitic stigmata were observed.

In the 45 cases where intravenous urography was performed, entirely normal conditions were found in 43. In two cases a slightly deformed pelvis was found on one side.

Among the 60 cases where the spinal cord was x-rayed, an incomplete closing or non-closing of one or more vertebrae in the lower lumbar or upper sacral region was found in 30 cases (50 per cent). In the control material (69 children who had not shown enuresis), spina bifida was observed in 34 cases or 49.3 per cent (see Hinze above).

In 47 cases, intelligence tests according to Binet-Simon or Terman were made; in other cases school reports and the follow-up examination had given some idea of the children's level of intelligence. Most of them were normally or exceptionally gifted, merely a small number of an auxiliary class standard, and two of them were inbeciles. Most of the children had been subjected at the hospital to the so-called Krasnogorski regime (restriction of liquid in the afternoon and a well-salted sandwich in the evening). In a few cases diuretin or phenemal-eumydrin was administered. Most of the children after discharge from the hospital were recommended a change of environment and were sent to a convalescent home for a month or longer.

During their stay in hospital most of the children had improved, some of them had even stopped wetting. The follow-up examination, however, showed that a considerable number were unimproved on returning home and that, as indicated by Table 19, p. 49, the relapses were frequent. Merely 7 per cent of the children had been freed from enuresis in direct connection with the hospital treatment, 38 per cent had improved, but nearly half of the latter had relapsed and more than half of the total number were unimproved after therapy. The results of the treatment were more favourable for girls than for boys (Table 20).

Table 20. Enuresis in boys and girls. Therapeutic results.

	Boys (75)		Girls (34)	
	Number	Percentage	Number	Percentage
Recovered	2	2.7 %	6	17.7 %
Improved	27	36 %	15	44.2 %
Relapses	12	44.5 %	7	46.7 %
Unimproved	46	61.3 %	13	38.1 %
At follow-up exam:				
easily educable	55	73.4 %	26	76.5 %
difficultly educable	20	26.6 %	8	23.5 %

If the results of the treatment are compared in relation to the duration of the change of environment, it appears that the lengthier changes had not given better results than the shorter.

The major part of the children had been subjected to the Krasnogorski regime at the hospital and most of them, when discharged, were advised to continue it. At the follow-up examination, however, it was found that only a small number of mothers had consistently carried out the regime for any length of time. What usually happened was that it was abandoned after a week or so, as the mothers had found it tiresome and had not noticed any effect. Only in 32 cases

was the regime carried out consistently for a month or longer. The results are shown in Table 21; they are not better than the general average (*cf.* Table 19 p. 49).

Table 21. Effect of Krasnogorski regime.

	Number	Percentage
Recovered	1	3.1 %
Improved	12	37.5 %
Relapses	6	50 %
Unimproved	19	59.4 %

In regard to the children treated with diuretin no conclusions can be drawn, there having been only two or three such cases. The group of children who received phenemal-eumydrin pills regularly for some length of time is likewise too small to admit of any valuation of the results.

Among small groups, it is, however, of interest to examine the results of the treatment in regard to children who had concurrently shown symptoms liable to cause or maintain the enuresis, notably those suffering from oxyuriasis or bacteriuria.

The three children who had been subjected to a worm cure for oxyuriasis had been completely and permanently freed from enuresis.

The two children suffering from bacteriuria had responded differently to sulfonamid medication. One of them had been directly freed from enuresis, which had not returned. The other continued wetting even after the urine had been freed from bacteria.

The part played by spina bifida in the etiology of enuresis has been much discussed, and it is therefore of interest to see whether children suffering from that complaint were more resistent to treatment than those without any defect in the lumbar vertebrae. (Table 22 p. 54.) Whereas among the children without spina bifida 41 per cent had recovered or had permanently improved after the hospital treatment, only 16.7 per

Table 22. Enuresis.

	With spina bifida	Without spina bifida	Σ
Recovered or improved (without relapses) . .	5 (n_{00})	12 (n_{10})	17
Unimproved	18 (n_{01})	13 (n_{11})	31
Σ	23	25	48

cent of those with spina bifida showed permanent improvement.

According to Dahlberg[1] the coefficient of correlation is

$$r_B = \frac{n_{00} \cdot n_{11} - n_{01} \cdot n_{10}}{\sqrt{(n_{00}+n_{10})(n_{01}+n_{11})(n_{00}+n_{01})(n_{10}+n_{11})}}.$$

The significance of the correlation is found with Fisher's transformation[2]

$$z = \frac{1^e}{2} \log \frac{1+r_B}{1-r_B},$$

with standard deviation

$$\varepsilon(z) = \frac{1}{\sqrt{N-3}},$$

where

$$N = n_{00} + n_{10} + n_{01} + n_{11}.$$

$$r_B = -0.2743$$

$$z = -0.2815$$

$$\varepsilon(z) = \quad 0.1491.$$

Thus z is not significantly different from zero and there exists no correlation between spina bifida and the therapeutic results in children with enuresis.

[1] Dahlberg, G., Statistical methods for medical and biological students. George Allen and Unwin, London, 1940.

[2] Fisher, R. A., Statistical methods for research workers. Oliver and Boyd, Edinburgh. 1938.

Table 23. Enuresis.[1]

	Day wetters 8 ch.	Day and night wetters 35 ch.	Night wetter 62 ch.
Recovered	1	3	5
Improved	4	13	22
Relapses	2	5	12
Unimproved	3	19	35
At time of follow-up exam. recovered	3	15	19
In conj. with *incont. alvi*	4	14	6

Table 23 shows the distribution of enuresis during the twentyfour hours. Most of the children wetted only at night, merely a few only in the daytime, several both day and night. In regard to therapeutic results, no marked difference between these groups could be observed. At the hospital, the night-wetters in more than half the cases had been over school age, the day-and-night-wetters in barely one-third of the cases over that age, most of the day-wetters under school age. In regard to the age at which the children had ceased wetting, corresponding conditions could be noted. The day-wetters had recovered first, next the day-and-night-wetters, who had first ceased day-wetting and then night-wetting. The night-wetters persisted longest, most of them, as it seemed, more or less regardless of possible attempts at therapy.

If we consider the date of the appearance of enuresis, we can distinguish two groups, namely the children who had wetted since infancy and those who had begun to do so after a free interval (Table 24 p. 56). Here, as shown by the Table, there is a distinct difference in the effect of the therapy, in that the children who had begun to wet after a free interval show a considerably smaller percentage of unimproved and a larger

[1] In regard to 4 children no information available.

Table 24. Enuresis.

After discharge	Enuresis since infancy (73 ch.)		Enuresis after dry interval (24 ch.)	
	Total	Percentage	Total	Percentage
Recovered	3	4.1 %	6	25 %
Improved (after fortnight) .	3		1	
» (» 1 month) .	3 } 24	32.9 %	1 } 12	50 %
» (» longer per.)	18		10	
Relapses	11	46 %	6	50 %
Unimproved	46	63 %	6	25 %

percentage of recovered and improved than those who had wetted since infancy. It thus seems that the prognosis is more favourable for children who had begun to wet after a dry interval, and that they are more amenable to therapy than those who had wetted since infancy. (Cf. Lichtenstein, above.)

As regards the children who, besides enuresis, had also shown incontinentia alvi, the enuresis had not been less amenable to treatment than in the others.

No difference in therapeutic effect could be observed between the backward children and those of normal intelligence, which seems to be due to the fact that only two imbeciles were included in the material. The other backward children were of the auxiliary class standard.

In the follow-up examination it was found that 70 children were still wetters. Several of them had reached the pre-puberal or puberal stage. 48 of them were in fact over 10 years of age, and out of the latter 16 were over 14 years.

Finally, if one examines more closely the eight cases who had recovered after the hospital treatment, one will find among them the three children suffering from oxyuriasis, who had recovered after a worm cure, as well as one of the children suffering from bacteriuria, namely an eight-year-old whose enuresis had persisted since infancy, but which ceased as soon as the urine, after sulfonamid medication, had been freed from

bacteria. Number 5 is a little girl of three and a half years, number 6 a little boy of three years, one month. The seventh child is a ten-year-old girl, who had wetted since infancy, but who had considerably improved even before the hospital treatment and was practically free from enuresis when she was admitted to the hospital. Finally, the eighth case is a five-year-old girl, who was sent to the hospital chiefly because of hysterical tantrums and had only wetted in bed quite occasionally. In this case the enuresis had thus been very mild and an accessory phenomenon of other nervous symptoms.

Summing up, it may thus be stated that in this material the therapeutic treatment had led to recovery where an organic cause of the enuresis was observed and where that cause (oxyuriasis, bacteriuria) had been removed; also in infants of about the age of three, in one case which was already on the way to recovery, and in one case of rarely occurring enuresis, where it was a transitory mode of reaction in an affect-labile child. No case of typical enuresis without an organic cause in children of school age was cured.

64 of the children affected with enuresis had come to the hospital with their parents for the follow-up examination, when character records were made. No special type of enuretici could be shown. Well-behaved and easily educable children were found among them together with those showing different behaviour problems. Friendly, open, accessible children occurred among them as well as tacit, reserved and oversensitive children. A strikingly large number, however, were timid, lively, motorially restless, affect-labile and easily tired. Most of them were stated to be normally matured and independent. Many of them (24 children) had restless sleep. As regards a considerable number, onanism had been more frequent than in other symptoms groups, apparently because the parents concentrated their attention on the bedwetters. No great frequency of other organoneuroses was observed in the bed-wetters. 13 of the children had had anorexia. Only 9 were stated to be soilers.

Enkopresis.

Enkopresis occurs firstly in imbecile and idiots, who never learn to be clean, secondly, though more rarely than enuresis, in children of normal intelligence. Enuresis and enkopresis often occur simultaneously, and the etiological factors mentioned in the literature are similar (see the section of enuresis). As an organic cause of enkopresis, a neglected constipation has often been found (LICHTENSTEIN in »Nordisk Lärobok i pediatrik»).

Among functional causes, sexual factors are considered to play a greater part in enkopresis than in enuresis (GLANZMANN, BENJAMIN, TRAMER), enkopresis being regarded as a manifestation of the »anal sexuality» mentioned by Freud. — Especially in children of school age, enkopresis may occur without enuresis. According to MARFAN, enkopresis solely is particularly common in pupils of the high school standard, who may even be exceptionally gifted and usually are affect-labile, impulsive and imaginative. TRAMER, on the other hand, has not found any special type of children affected with enkopresis.

Own material.

Normal children usually learn at a considerably earlier stage voluntarily to control their evacuation than the voiding of their bladders.

Though it is usually expected that a child shall voluntarily control evacuation after reaching the age of two, the age limit has been set here at three years, as applies also to voluntary control of the voiding of the bladder (see above). Those children who at the time of the hospital treatment were under three have been regarded as affected with enkopresis when that symptom had not ceased after reaching that age.

In 18 cases enkopresis was one of the symptoms for which the children had been hospitalized; among them merely two were girls, the others boys. In all cases but one, enkopresis occurred in conjunction with enuresis.

In addition to the routine physical examination, which revealed nothing special, and the investigations occasioned by enuresis, rectal palpation was made in all cases and signs of constipation were looked for. In all cases the sphincter was normal, without signs of relaxation. In two cases the children had obstipation. X-ray examination of the colon was made in five cases, all of which were negative. One child had oxyuriasis. In four cases the lumbar vertebrae were x-rayed and in two of them spina bifida occulta was observed.

Intelligence tests were made in 10 cases, whilst in others information from the school, or specimen tests at the follow-up examination, made it possible to form an idea of the children's intelligence level. 16 of them were of normal intelligence, one of them of the auxiliary class standard and one an imbecile.

In the two cases suffering from obstipation, the therapy was adapted accordingly; the child suffering from oxyuriasis was subjected to a worm cure, in the other cases the treatment consisted solely in a change of environment.

Table 25. Enkopresis.

Age on admission	2—3 years	3—4 years	4—7 years	7—8 years	8—9 years	9—10 years	Total	%
Number of children . . .	3	5	5	1	1	3	18[1]	
After discharge:								
Recovered		1	3		1	2	7	38.7
Improved		3	2	1			6	33.3
Relapses				1			1	16.6
Unimproved	3	1				1	5	28

[1] 16 boys, 2 girls.

The results are shown in Table 25, where all the cases are included. As will be seen from the table, a good many children had recovered directly after the hospital treatment and several of them had improved. Among the improved cases merely one child had relapsed. The unimproved cases (among

which we find the smallest children and the imbecile) amounted to 28 per cent. Among the improved, the child of auxiliary class standard was the only case of relapse.

Table 26. Enkopresis.

Age	3—4 years	4—7 years	7—8 years	8—9 years	9—10 years	10—12 years	12—14 years	14—16 years	Total
Age at time of recovery . .	2	3	2	1	3				11[1]
Continued enkopresis at time of follow-up examination		1	1		1	1		1	5

[1] As regards 2 children no information available.

The age of the children who still showed enkopresis at the time of the subsequent examination is seen in Table 26. In the last-mentioned group we find the imbecile and the auxiliary-class type of child, the former being 6 years old, the latter 11 at the time of the examination.

The cases are too few to allow classification in small groups, according to sex, the duration of the change of environment, and so on. No special type could be observed. At the time of the follow-up examination two-thirds of the children were found to be easily educable.

Table 27. Comparison between therapeutic results in children with enuresis and enkopresis.

	Enuresis	Enkopresis
Recovered	7.8 %	38.7 %
Improved	38.6 %	33.3 %
Relapses	45.5 %	16.6 %
Unimproved	54.1 %	28 %

In comparing the therapeutic results in cases of enkopresis and enuresis (Table 27), it will be found that enkopresis responded considerably better to treatment. Generally speaking,

enkropresis ceased at an earlier date than enuresis. That the prognosis for enuresis in conjunction with enkopresis was not found to be unfavourable, has already been indicated (page 56).

Symptoms from the nervous system.

Symptoms from the nervous system are fairly common in nervous children. In the following only those symptoms from the nervous system which are represented in the hospital material will be mentioned.

1) *Headache*, which is an extremely common nervous symptom in children, may take the form either of ordinary headache, or of migraine. HOLT found headache chiefly in girls between the ages of ten and fourteen. According to TRAMER, headache often occurs in conjunction with fatigue, a tendency to isolation, a marked feeling of discomfort, disorders of sleep, anorexia, etc. According to BENJAMIN, migraine in the age of childhood often has an atypical course, *i. e.* is frequently not unilateral and not accompanied by eye symptoms or vomiting. HOLT too has very rarely found typical migraine in children. Some authors consider that umbilical colic is closely associated with migraine. BENJAMIN, however, contends that the amenability of umbilical colic to suggestion and its tendency to disappear on a change of environment argue against such a connection.

2) *Tic* signifies stereoptyped, voluntary spasmodic movements, occurring at certain intervals, and often rhythmical, which, though originally purposive, attract attention by their exaggerated character (BENJAMIN-HOLT). There is a close connection between tic and bad habits, and HOMBURGER notes the occurrence of indeterminate transitions between those phenomena. In BENJAMIN'S material 70 per cent of the children suffering from tic are of the regression type, only a few of the stubborn type, whilst 20 per cent are »Abwendungstypen».

3) *Convulsions* of a functional nature occur partly as breath-holding spells usually between the ages of 1 and 5 years, partly

as hysterical attacks of a different nature in older children. BENJAMIN includes the breath-holding spells among the typical negativistic reactions, but does not place the hysterical attacks in any special group. According to LINDBERG's classification, children suffering from convulsions are to be regarded as sub-solid.

4) *Disorders of sleep* of various types, such as restless sleep, pavor nocturnus, somnambulism, etc. occur. Setting aside those disorders of sleep which are due to organic causes, they are often symptoms of anguish (BENJAMIN). BENJAMIN also points out that few nervous symptoms are so amenable to treatment with a change of environment as disorders of sleep. He points out also that, besides anguish, the desire to attract the attention of those around them is a prominent factor in various disorders of sleep, thus explaining the favourable effect of change of environment. LICHTENSTEIN likewise points out that in many cases such disorders may be attributed to faults of education. According to TRAMER, restless sleep is due to conflict situations which the child is unable to cope with. It is also interesting to note that, according to him, certain cases of restless sleep and pavor nocturnus may be due to lack of glycogen and can be cured by the administration of sugar. LINDBERG considers that pavor nocturnus and somnambulism may be included in the sub-solid symptom group.

Own material.

Tic.

This group includes children who had shown involuntary muscular twitches, where febrile diseases, such as encephalitis or chorea, were not found in the anamnesis, and where chorea was definitely excluded by clinical observation.

At the follow-up examination it was found that 29 of the children (18 boys and 11 girls) were suffering from tic. The age-distribution at the time of the hospital treatment is shown in Table 28 p. 63. Most of them were of school age. In 19 cases

Table 28. Effect of treatment of children with tic.

Age group	3—4 years	4—7 years	7—12 years	12—15 years	Total
Onset at age of	1	6	21	1	29
Recovered after hospital treatment	—	3	3	—	6 = 20.7 %
Improved » » »	—	3	8	1	12 = 41.4 %
Unimproved » » »	1	1	9	—	11 = 37.9 %
Relapses » » »	—	1	1	—	2 = 16.2 %

the tic was rather widespread, being usually located in the upper extremities, in some cases also in the lower. Others had a more localized tic, for example in the form of blinking, snorting, hawking or the like. The anamnesis shows that all the children but one also had other nervous symptoms, such as disorders of sleep, anorexia, abdominal pains, motorial unrest, tantrums, etc.

One of them had been prematurely born, two others were retarded in development. In some of them systolic murmurs (merely in one case indicative of vitium cordis) had been heard.

Most of them had shown rather exaggerated reflexes; otherwise no pathological phenomenon had been observed in the neurological examination.

Intelligence tests were made in 19 cases; in regard to the others, information obtained from the school or at the follow-up examination was available. One of the children was mentally defective, another of the auxiliary-class standard, the others of normal intelligence.

As regards hereditary factors, 50 per cent of the children had a nervous taint. The enviroment showed nothing of particular interest. In certain cases the home conditions seemed to be rather bad, but in no greater degree than in other grops.

The therapy consisted chiefly in the change of environment. In some cases treatment at a convalescent home was recommended. In most cases phenemal was administered at the hospital and prescribed on discharge.

The effect of the treatment is shown in Table 28 p. 63. Several of the children had recovered directly after the hospital treatment (20.7 per cent), but many were unimproved (37.9 per cent). Out of the improved, merely two had relapsed. A lengthy change of environment had no better effect than a shorter one. Widely distributed tics were quite as amenable to treatment as tics localized in a limited area, such as blinking tic. The results in fact indicate that localized tic was more resistent to therapy. The groups, however, are rather small.

Improvements at different ages were observed. In some cases the tic had disappeared at an early school age, in others at puberty. Out of those who still had tic (8 children) at the time of the follow-up examination, only one girl had passed the puberal stage.

When the characteristic data were surveyed, it was found that about 50 per cent of these children were awkward, reserved, tacit, introvert, unsociable, with merely a few friends. About as many had disorders of sleep of differents kinds, several of them pavor nocturnus. Most of them were nervous, sensitive and delicate. At the follow-up examination most of them (79.4 %) were stated to be easily educable, though many were still rather nervous and sensitive. Among the six problem children we find the imbecile child (see above).

Headache.

9 of the children had had headaches without any observable organic cause. The cases are too few to warrant further comment.

All of them were of school age, two-thirds of them being girls, the other third boys. The physical examination showed nothing of interest. In all cases the headaches had been occasioned by school difficulties, though the intellectual qualifications, in all cases but one, were good: some of the children were even exceptionally gifted. In a single case the headache was unilateral, but without vomiting or symptoms from the eyes. In the other cases it was bilateral, in one of those ac-

companied by vomiting and ophthalmic symptoms. In one case the mother had migraine; otherwise there was no hereditary disposition for that complaint, though in a few cases for nervousness.

The environment was bad in two cases, one of which was an adoptive child. In all cases the therapy consisted in a change of environment and attempts to better the school situation, for example by transfer to another school; in other cases endeavours were made to settle the school conflict by talks with the teacher.

The results of the therapy are shown in Table 29. The cases are too few to warrant any conclusions. Seven of the

Table 29. Headache (3 boys, 6 girls).

	7—12 years
	Number of children
Recovered	1
Improved	5
Relapses	2
Unimproved	3

children were characterized as sensitive, unsociable, reserved, nervous and affect-labile and had few playmates. At the time of the follow-up examination two-thirds of the children were found to be easily educable.

Disorders of sleep.

An extremely common symptom in all the children was some kind of disorder of sleep. This group comprises only the 40 children who had been hospitalized partly for that reason.

The age-distribution is shown in Table 30 p. 66. In the physical examination no organic cause of the disorder could be observed, but all the children showed other nervous symptoms.

Table 30. Disorders of sleep.

Age at time of hospital treatment	0—1	1—2	2—3	3—4	4—7	7—12	12—15 years	Total	%
Number	2	7	7	5	8	9	—	40	
Completely recovered after hospital treatment	1	3	2	1	1	1	—	9	22.5 %
Improved (more than 1 month) after hospital treatment	—	3	2	3	4	5	—	17	42.5 %
Unimproved after hospital treatment	1	1	3	2	4	3	—	14	35 %
Relapses after hospital treatment .	1	1	—	1	2	1	—	6	35.3 %

The different disorders of sleep are tabulated in Table 31, which also shows that the nature of the disturbance varies with age. A sudden and more or less incomplete, anxious awakening was designated as pavor nocturnus, whereas a quiet awakening following by screaming was classed as screaminess. At the age of 1—4 years the night terrors and screaming predominate; at more advanced ages the children had slept restlessly, talked in their sleep and in a few cases had shown real somnambulism.

The therapy consisted almost solely in a change of environment; in some cases treatment in a convalescent home was

Table 31. Different disorders of sleep.

	Age distribution					Results		
	0—1	1—4	4—7	7—12	12—15 years	Recd.	Imp.	Unimp.
Night terrors	—	9	2	1	—	4	5	3
Restless sleep, dreams	—	2	5	5	—	2	4	6
Difficulty in falling asleep	—	3	1	2	—	—	4	2
Awakes in middle of night or very early	—	2	—	1	—	1	1	1
Screams	2	2	—	—	—	1	1	2
Talks in sleep	—	—	—	2	—	—	2	—
Somnambulism	—	—	—	2	—	1	1	—
Jactatio caputis	—	1	1	—	—	—	1	1

recommended. In a few cases soporifics were given on isolated occasions, such as the first night at the hospital, but not afterwards.

The therapeutic results are shown in Table 30 p. 66. About one-fourth of the children had recovered directly after the hospital treatment and over 40 per cent had improved. Many, however, had relapsed. The most satisfactory results were attained between the ages of 0—4 years (33 % recovered) as compared with the older children (11.6 % recovered in the group 4—12 years), as night terrors had readily responded to treatment. Table 39 indicates that 75 per cent of the children suffering from night terrors had permanently recovered or improved after treatment in the hospital.

In 58 per cent of the children disorders of sleep occurred in conjunction with other organoneuroses, as a rule with anorexia and umbilical colic, but also enuresis or tic. In such cases the disorder of sleep was not found to be less amenable to treatment than if it had been the sole nervous symptom.

No further classification into groups, *e. g.* according to the duration of the change of environment, was considered necessary, as such groups would have been unduly small.

In the character analysis, it was not found that any particular type was predominant.

At the time of the subsequent examination 62.5 per cent were stated to be easily educable.

Convulsions.

This group comprises firstly children with breath-holding spells, secondly those who had had attacks of a hysterical nature. In all cases physical examination indicated that the attacks had no organic basis, nor was there reason to suspect endogenous epilepsy. The group is quite small, comprising merely 11 children (4 boys and 7 girls). Six of the children were under the age of four and had had breath-holding spells, the other five were of school age and had had attacks of a hysterical character.

Table 32. Convulsions.

Age at time of hospital treatment	0—1	1—2	2—3	3—4	4—7	7—12	12—15 years	Total	%
Number	—	2	3	1	—	5	—	11[1]	
Recovered	—	1	2	1	—	5	—	9	81.9 %
Improved	—	1	1	—	—	—	—	2	18.1 %
Relapses	—	—	—	—	—	—	—	—	—
Unimproved	—	—	—	—	—	—	—	—	—

[1] (4 boys + 7 girls).

The treatment consisted in a change of environment and pedagogic advice. The results of the treatment are shown in table 32. Most of them had not had any attack after discharge from the hospital; only two of the children had had a few mild attacks after returning home, but otherwise were permanently improved. In this group there are no relapses.

At the time of the follow-up examination all the children were stated to be easily educable. The cases are too few to permit any attempts at character analysis.

Summary of the therapeutic results.

The therapeutic results in all the symptom groups are summarized in Table 33, p. 69.

If the immediate results of the therapy in (1) children treated for behaviour problems and (2) children with various organoneurotic symptoms are compared, it will be seen from the table that the organoneurotic symptoms were not more amenable to therapy than the behaviour problems. Though the percentage of complete recoveries is somewhat higher in children with organic neuroses than in those with behaviour problems, the number of improved is smaller in the former and the number of unimproved larger. It has been found that anorexia and enuresis are symptoms which are particularly unamenable to treatment. In these symptom groups about half the number of children were unimproved after treatment.

Table 33. Summary of results at follow-up examination of 337 children treated at Kronprinsessan Lovisa's hospital for various nervous symptoms.

Symptoms	Number	After hospital treatment				At follow-up examination		
		Recovered	Impr.	Relapses	Unimpr.	Mild cases	Severe cases	Mentally diseased
Behaviour problems	175	10 %	64 %	36 %	26 %	59 %	41 %	1.2 %
Organic neuroses:								
Anorexia	45	15 %	38 %	33 %	47 %	*50* %	*50* %	
Umbilical colic and vomiting	53	49 %	40 %	30 %	11 %	76 %	24 %	
Heart troubles . . .	5	60 %			40 %	60 %	20 %	20 %
Enuresis	109	7 %	39 %	46 %	54 %	74 %	26 %	
Enkopresis	18	39 %	33 %	17 %	28 %	63 %	*37* %	
Tic	29	21 %	41 %	16 %	38 %	79 %	21 %	
Headache	9	11 %	56 %	40 %	33 %	63 %	37 %	
Disorders of sleep .	40	23 %	43 %	35 %	34 %	63 %	*37* %	
Attacks (breathholding spells or hysteria)	11	82 %	18 %			100 %	—	

and within these groups relapses were specially frequent. Setting aside the groups with heart troubles and convulsions (within which the cases are too few to warrant any comparison), the best results were attained with children suffering from umbilical colic and vomitings, most of whom had recovered or improved.

If, on the other hand, we consider the children's further development, quite different conditions will be brought to light. Children with organoneurotic symptoms showed, in the sequel, fewer behaviour problems than those who had been treated for difficulties of a purely pedagogic character. Exception must, however, be made for children with anorexia, a very high percentage of whom were found at the follow-up examination to be difficultly educable. In fact, enuresis and anorexia were the organoneurotic symptoms that were most re-

sistant to therapy (see above). On the other hand, most of the children with enuresis, unlike those with anorexia, were found at the follow-up examination to be easily educable.

General summary.

The investigation comprises 337 children who in 1935—1939 had been treated at Kronprinsessan Lovisa's Children's Hospital in Stockholm, and who were subjected to a follow-up examination in the autum of 1941.

Proceeding from the particular symptom for which the child had been sent to hospital, different symptom groups were drawn up. The material was divided into two main groups, namely (1) children showing behaviour problems and (2) children with organic neuroses.

Behaviour problems.

175 children, being 109 boys and 66 girls, showed behaviour problems as dominant symptoms.

A hereditary disposition for mental disease, psychopathy and »nervousness» was found with much greater frequency in these children (56.9 %) than in a control material of organically diseased children without nervous symptoms (20 %).

The percentage of the children who had come from defective homes was considerably larger in the problem children (47.6 %) than in the above-mentioned control material (20 %).

Many of the children showed organoneurotic symptoms as well as behaviour problems.

Most of the children were of normal intelligence.

Five different symptom groups were drawn up, namely (1) screaming, hot-tempered, stubborn children, (2) motorially restless children, (3) sulky and callous children, (4) delicate, languid, tired, peevish children, (5) hypersensitive, restless and nervous children.

The therapy consisted in pedagogical advice, attempts at improvement of the home conditions and change of environment.

Therapeutic results.

Out of the total number of children, 10.3 % had recovered in immediate connection with the treatment, 64 % had improved, and 25.7 % had remained unimproved after therapy, 36.3 % of the improved had relapsed.

The percentage of recoveries after treatment was largest in children under the age of three years, and fell with advancing age.

The symptoms which were least amenable to treatment were sulkiness and callousness, which also showed the highest percentage of lasting behaviour problems (87.5 %).

The group which showed the next largest percentage of children with lasting behaviour problems was found to be that of the stubborn children.

A comparison was made between children who, at the follow-up examination, had passed the puberal stage and those who were still younger. It was found that the percentage of problem children among the former was considerably lower than among the latter (27.9 and 49.6 %, respectively).

In 1.2 per cent of the children schizophrenia had developed.

The behaviour problems were grouped according to degree of severity. Many of the children who during the hospital treatment had shown serious behaviour problems had afterwards become amenable to education (37 %). This indicates that even grave behaviour problems may be due to maladjustment. For this reason, great caution should be shown in diagnosing psychopathy at the age of childhood.

Certain symptoms, such as vagabondage, destructiveness and cruelty to animals are indicative of an unfavourable social prognosis.

In children who, besides behaviour problems, had shown organoneurotic symptoms, the therapeutic results were not worse than in those without such symptoms.

Problem children with a hereditary predisposition for nervous diseases were amenable to therapy in the same degree as children without such hereditary taint, but among the

former the number of relapses after a short time was about twice as large as among the latter. Moreover, a considerably larger number of children with a hereditary predisposition for nervous diseases were found at the follow-up examination to be difficult to educate than those without such hereditary taint.

Children from defective homes recovered and improved to the same extent as those who had been brought up in normal home conditions. This indicates that in such cases there are good prospects of improving the environment.

Organic neurosis.

Anorexia. This group comprises 63 children, who were divided into two sub-groups, namely (1) children with so-called genuine anorexia and (2) children with secondary anorexia (where an immediate organic cause of the anorexia could be shown).

Genuine anorexia usually occurred during the first year of life, and, with a few exceptions, not later than during the age of 2—3 years.

A certain hereditary predisposition for nervous diseases was shown in 43 per cent of these children.

The family conditions were found to be a factor of considerable importance. In many cases it appeared that a child affected with genuine anorexia was a first or only child.

Anorexia responded with difficulty to therapeutic measures. Genuine anorexia was found to be less amenable to therapy than secondary anorexia.

Lengthy changes of environment appeared to have somewhat better effect than shorter ones.

Anorexia usually cleared spontaneously at or before the age of seven.

Many of the children with anorexia were found to be difficult to educate during the period of growth, and several of them presented serious behaviour problems.

A large number of these children were designated as independent, affect-labile, stubborn and tyrannical.

Umbilibal colic. This group consists of 38 children, most of them of school age. A considerable number of them (26.3 %) showed a certain hereditary predisposition for nervous diseases. In about 50 per cent of these cases the abdominal symptoms had been precipated by school difficulties.

The results of therapeutic treatment were relatively favourable, about half the number of these children having immediately recovered.

A large percentage of the children with umbilical colic were found at the follow-up examination to be easily educable (79 %).

Those suffering from umbilical colic were usually lively, nervous, sensitive and affect-labile children, delicate in constitution, who reacted with abdominal pain to mental strain.

Vomiting. This group comprises 15 children, most of them of school age.

A hereditary predisposition for nervous diseases was found in about 40 per cent of these children. — In all the school children the vomiting had been precipitated by school difficulties.

The results of therapeutic treatment were relatively satisfactory: many of the children had immediately recovered (40 %). These children were found to be of the same type as those suffering from umbilical colic (see above).

Nervous heart symptoms occurred merely in five cases, which are dealt with very briefly. (See Table 18, p. 46.)

Enuresis. 109 children with enuresis were followed up.

A hereditary predisposition for nervous diseases was found in a large number of these children, and in many cases enuresis had occurred in the family. Malformations or morbid processes in the external genitalia were found merely in an extremely small number of cases, and no signs of syphilis were observed.

The therapeutic results were unsatisfactory.

Lengthy changes of environment had no better effect than shorter ones.

The Krasnogorsky diet was not found to be of any avail.

Children suffering from oxyuriasis and bacterieuria were found to be amenable to therapeutic treatment. (These cases, however, were too few to permit any general conclusions). The occurrence of spina bifida occulta was quite as frequent in children with enuresis as in those in a control material without enuresis.

No correlation existed between spina bifida and therapeutic results in children with enuresis.

Children who had begun to wet after a dry interval were found to be much easier to deal with than those who had wetted since infancy.

No special type of enuretici could be observed.

Enkopresis. This group comprises 18 cases. In all cases but one it was found in conjunction with enuresis. The therapeutic results were more favourable than as regards enuresis. In the character analysis no special type of child predominated. Two-thirds of the children were found at the follow-up examination to be easily educated.

Headache, tic, disorders of sleep and convulsions.

9 children, all of school age, had had *headache* as a dominant symptom. In all cases the headache had been precipitated by school difficulties. The cases are too few to warrant any conclusions.

29 children, most of them of school age, showed *tic.* A hereditary predisposition for nervous diseases was found in about 50 per cent of them.

The results of the treatment were rather unsatisfactory, with a fairly large percentage of unimproved (37.9 %).

Lengthy changes of environment had no better effect than shorter ones.

Widely distributed tic was quite as amenable to treatment as tic localized in a smaller region.

Half of the children with tic were awkward, reserved, tacit introverted and solitary. At the follow-up examination most of them were found to be easily educable.

38 children showed *disorders of sleep* as dominating symptoms. The nature of the disorder varied with age.

The therapy, which consisted mainly in a change of environment, had a fairly good effect, but the relapses were numerous. The best results were attained with the treatment of pavor nocturnus.

At the follow-up examination more than half the children (62.5 %) were found to be easily educable.

Children with functional *convulsions* were few in number and, though children with other attacks of a hysterical nature were included in this group, it comprises merely 11 children. In all cases the therapeutic results were satisfactory, and at the follow-up examination these children were found to be easily educable.

A comparison between the therapeutic results in different symptom groups shows the following: —

Organoneurotic symptoms and behaviour problems were almost immediately affected, in approximately the same degree, by therapeutic measures. At the follow-up examination the children who had suffered from organic neuroses were found to be easily educable in a larger percentage than children who had chiefly shown behaviour problems. An exception must be made as regards children with anorexia, many of whom were found at the follow-up examination to be difficult to educate.

References.

AHNSJÖ, Act. pæd. Vol. XXVIII, suppl. 3, 1941. BENDER and YARNELL: Am. J. Psychiat. 97, 1941. — BENJAMIN, HANSELMANN, ISSERLIN, LUTZ and RONALD: Lehrbuch der Psychopathologie des Kindersalters. Rotapfel, Zürich, 1938. — BENJAMIN: Ztschr. f. Kinderpsych. 6: 161—169, 1940. — BÜHLER: Kinderheit und Jugend. — GRÜNEBERG: Annales paediat. 152: 178—191, 1938—1939. — BOAS: Diagnostik und Te-

rapi der Magenkrankheit. Leipzig 1920. — FAIRBANK: Mental Hygien 17: 177—208, 1933. — GRAFE: Dtsch. Med. Wchnschr. 39, 1939. — HINTZE: Dermat. Wchnschr. 385, 1942. — HOLT, MC INTOSH: Diseases of Infancy and Childhood. Appleton Company, New York, London 1933. — KANNER: Am. J. Psychiat. 13: 171—182, 1933. — —: Child Psychiatry, Springfield 1935. — —: Am. J. Psychiat. 94: 691—699, 1937. — —: Am. J. Psychiat. 97: 2, 1941. — KARGER: Dtsch. Med. Wchnschr. 20: 639, 1924. — KISTINCENKO: Dermat. Wchnschr. 112: 85—92, 1941. — LANGE: Fortschritte d. Neurol. Psychiat. 12, 101 und 121, 1940. — LICHTENSTEIN: Nordisk lärobok i pediatrik, Munksgaard, Köpenhamn 1941, Bd. 2, 337—350. — LINDBERG: Nordisk Medicin. Bd. 19, 1475, 1943. — LURIE: Am. J. Psychiat. 91: 1379—1388, 1935. — NEURATH: Die Pubertät. Springer 1932. — NYCANDER: Nordisk Medicin 1: 115—121, 1939. PINARD: L'énurésie, son étiologie. Paris. Med. 9, 1934. — POTTER, KLEIN: Am. J. Psychiat. 94: 1, 681, 1937. — RICHARDS: J. paediat. 18: 528—537, 1941. — SCHRÖDER: Ztschr. ärtzl. Fortbild. 36: 196—199, 1939. — SCHWALBE-HANSEN: Hospitalstiden. 81: 407—415, 26: 1938. — TRAMER: Kinderpsychiatrie, Benno Schwabe & Co, Basel, 1942. — —: Ztschr. f. Kinderpsychiatrie, 7: 77—81, 1940.

DELINQUENCY AND CHILDREN
FROM BAD HOMES

FROM THE STATE INSTITUTE OF HUMAN GENETICS AND RACE BIOLOGY, UPPSALA
(HEAD: PROFESSOR GUNNAR DAHLBERG M.D., LL.D.)

DELINQUENCY AND CHILDREN FROM BAD HOMES

A STUDY OF THEIR PROGNOSIS FROM A SOCIAL POINT OF VIEW

BY

EDITH OTTERSTRÖM

ACTA PAEDIATRICA VOL. XXXIII SUPPLEMENTUM V

STOCKHOLM 1946

LUND 1946
CARL BLOMS BOKTRYCKERI A.-B.

TO THE MEMORY
OF MY PARENTS

PREFACE

For a long time now there have been Child Welfare Boards in Sweden that among other duties have to take care of delinquent children and children from bad homes who are in danger of becoming delinquent.

The present work is intended to give a survey of the home conditions and other characteristics of such children, and above all to show how the children turn out in later life. The material under investigation consists of 2 346 children, 1 727 boys and 619 girls. Most of them (1 601) have been taken in charge for protective education by the Child Welfare Board in Malmö — the third largest town in Sweden —, but the material also consists of youths (745) who have been subjects of Court measures.

The investigation was commenced during the autumn of 1942 but did not assume a more definite form until 1943, after I had come into contact with Professor GUNNAR DAHLBERG, Chief of the State Institute of Human Genetics. My plan of work proved to fit in well with the programme of the Institute and with the series of investigations which it is making into delinquent and mentally retarded persons. I therefore had the privilege of consulting him, and was aided by the Institute in the collection of certain data. When in due course a costly treatment of the material was necessary, it was of tremendous advantage for me to have this done free of charge at the Institute and to avail myself of its stores of investigations and records in social medicine and experience of statistical treatment of materials. For this as also for his kindness in giving me place of work at the Institute I am very much indebted to Professor Dahlberg. Furthermore, I should like in particular to thank him for his never failing interest and inspiring guidance.

My hearty thanks are due to the Staff of the Institute, in the first place Aktuarie ELVIR LANDER, for the valuable help I received with the statistical treatment. I should also like to offer warm thanks to Miss MARGIT SKATTMAN, who carried out the actual work of calculation. I have furthermore received kind assistance from Miss SAGA NEANDER, Mrs ELLA TIBBLING and Miss METTA LINDBERG.

To Kyrkoherde HAGBARD ISBERG, one of the pioneers of the social care of children, I would tender my warm gratitude for the opportunities he has given me of availing myself of his experience from the earliest years of the activity of the Child Welfare Board and especially for the encouraging interest he displaced during the first troublesome steps in my work.

I am particularly indebted to Rådman C. G. NANNE, formerly Chairman of Malmö Child Welfare Board, for permission to inspect the records of the Board and for his constant interest while the work was in progress.

My cordial gratitude goes to the Assistant at the Child Welfare Board in Malmö, Miss HILDUR ANDERSSON, who gave up her free time to help me with the collection and supplementation of primary data.

I should like to thank the following Heads of different public authorities in Malmö for various items of information supplied to me: Assessor N. ARASKOG, Director of the Public Census Office, Mr E. PALLIN, Director of the Poor Relief Board, Mr A. SJÖHAGEN, Director of the Temperance Board, Folkskolekamrer A. WILLERT, and Mr E. KASTENGREN, Director of the Central Prison in Malmö. My thanks are also extended to their staffs.

I am also indebted to the Director of the Penal Register, Byrådirektör A. L. BERGGREN, and Förste Aktuarie HANS GAHN of the Royal Control Board (Kungl. Kontrollstyrelsen) for their kindness in providing information.

My warm thanks are also proffered to Stadsfogde RAGNAR LAURIN, Chairman of Landskrona Child-Welfare Board, for information as to the practical application of the Child Welfare Laws.

I am further greatly indebted to vicars and staffs at the parish

registry offices in Malmö and in a number of places about the country for their kind help in tracing all those individuals in the material who proved difficult to follow on account of constant removals.

It has been possible to print the work thanks to a grant from the Hormone Laboratory of the Women's Clinic of the Public Hospital in Malmö. I should like to offer my warm thanks to the Head of the Laboratory Med. Dr SUNE GENELL, for his assistance.

The translation has been done by my colleague Med. Dr ROLF HALLGREN and Mrs CAROLYN KING to whom I tender my sincere thanks.

I should also like to thank the officials of the City Library in Malmö for all ready help and kindness they have shown me in procuring the literature.

Upsala 11 Oct., 1946.

Edith Otterström.

CHAPTER I

SURVEY OF LITERATURE

Different theories have been launched to explain the problem of juvenile delinquency. Without for the moment discussing at length the literature dealing with these, it may be pointed out that delinquency may be either of genetic origin or the effect of environmental influences. Theories vary as to the relative importance of these two factors. Some authors hold heredity mainly responsible, while others are inclined to ascribe a decisive importance to environment. This issue, of course, can hardly be decided by a priori discussion. At present it is above all necessary to collect factual evidence from which general conclusions may subsequently be drawn. Conditions vary in such a way that in one case heredity must be considered to predominate whereas in another instance environment appears to be obviously decisive.

In addition, conditions are not the same in different countries or at different periods of time. Environment presumably plays a more important rôle among slum dwellers than in a population living under less unsatisfactory conditions. Furthermore corrective action against delinquency is by no means decided on uniform lines. Only recently has the idea become generally accepted that the community ought to intervene with delinquent children, a circumstance which is reflected in rising figures for delinquency. It is also obvious that parents of good social standing are better able to protect their delinquent child from corrective measures by the civic authorities, being in a position to make private arrangements for its care.

As a result of all this the conclusions reached by different

investigators frequently disagree and what is more, are of a nature to obviate comparison.

These reservations must be kept in mind when endeavouring to elicit from the literature the current opinions on delinquency.

FREQUENCY.

The frequency of delinquency obviously depends on what we mean by this term. The concept is variously interpreted in different countries and at different times. An estimate of frequency at an earlier date is accordingly hard to deduce. Only recently, in connection with the promulgation of special enactments for the care of children, have statistical data been registered concerning such delinquent juveniles as have been taken in charge by the Child Welfare Board. Just as criminal statistics, however, leave a margin between legal and real criminality, so does a corresponding discrepancy exist between the true number of delinquent children and the number of juvenile delinquents who have become known to the authorities.

The countries where the principles of protective legislation and the general structure of society are most akin to ours are the other Scandinavian ones. Norway was the pioneer country in this field. Protective legislation was introduced in that country already in 1896, but was not enforced until 1900. In Sweden the first law »*Education of delinquent and morally neglected children*» dates from June 13th 1902. This act was superseded on June 6th 1924 by »*the Child Welfare Law*» which since 1934, when the age limit of the persons covered by the act was in certain cases extended to 21 years, is denominated »*Law regarding Child Welfare and Juvenile Protection*». In Denmark the first law protecting children was passed in 1905, and in Finland on Jan. 1st 1937.

The earliest statistical accounts covering the activities of the Child Welfare Board in Sweden date from 1928.

The figures show that the number of children submitted to protective education in the entire country was fairly constant from 1928 to 1937 inclusive. During the years 1938—1940 a

Table 1. **Number of children under 18 years of age submitted to protective education.**

From »Sveriges officiella Statistik» (Official Statistics of Sweden), Samhällets Barnavård.

Year	1928	1931	1934	1937	1940	1943
Number ...	1 061	1 049	938	906	950	1 341

slight increase of about 5 per cent is noted, but during the following period to 1943 an increase of more than 40 per cent arose. Direct variations in the frequency of delinquency, however, cannot be deduced from these figures, because action in some instances has been taken for reasons other than delinquency. Thus, children are included who were taken in charge because they had been subjected to neglect or cruelty in their homes (Paragraph 22, clause a), as also children whose home conditions were bad generally (Paragraph 22, clause b.) It is furthermore obvious that the relative number of these children is greater in the towns than in the country. On the whole, protective education has to be resorted to twice as often in the towns as in the country (8 against 4 per thousand).

No investigation into the frequency of delinquency in boys has been carried out in Sweden. AHNSJÖ (1941) in an inquiry into girl delinquency was able to show that 0.2 per cent of all girls born in Sweden had been in detention homes or reformatories. The figure for the country was 0.1 per cent, that for the towns 0.4 per cent. There was a tendency towards increase in rural districts, whereas a certain stability seems to have been attained in the towns.

According to earlier current opinion the girls form a very small part of the delinquency clientele. This view was founded on the relatively low percentages of girls reported by preceding authors. The apparent difference between the sexes might be due to a lesser degree of delinquency in girls, which could be dealt with by methods less rigorous than detention, other arrangements being substituted. The girls' share in the contingent of the delinquents is also dependent on the extent to which sexual misbehaviour, which plays such an important part

where girls are concerned, is brought before the Child Welfare Board. This kind of misdemeanour is viewed with different eyes in different places. According to the official statistics quoted above, the sex distribution among the juveniles under 18 years who had been taken in charge has always shown the boys to be in the majority. During 1940 the figures were 56.7 and 43.3 per cent respectively, and in an official report from the years 1941 and 1942 made by JONSSON (1944) the number of girls was roughly one third of the number of boys (1 061 girls against 3 352 boys).

Naturally delinquency and juvenile criminality only coincide to a certain extent. Delinquency is the broader concept and refers mainly to lower ages. Juvenile criminality refers to varying ages in different countries, the age limits for legal responsibility being different. Criminality presupposes a definite and more or less spectacular criminal act. We speak of delinquency even in cases where there is no question of more palpable misdemeanour. Delinquency accordingly constitutes a wider concept. It might, however, be of interest to review briefly the literature on juvenile criminality also.

Towards the end of the 19th century juvenile criminality was on the increase in most European countries as well as in Sweden. It is, however, impossible to obtain comparable tabulated surveys on this subject. Swedish conditions in this respect for the years 1909—1914 are submitted in Poor Law Report (1915) (»Fattigvårdslagstiftningens betänkande»). The figures are given in Table 2. Percentages have been calculated for the boys, whereas the girls are so few as to render such treatment pointless.

The figures do not appear to be high, — on the contrary they are unexpectedly low. They only refer to persons who have been sentenced to jail or penal servitude. It is remarkable that these figures do not indicate a substantial increase in this category.

Investigation into juvenile criminality by the Central Prison Board (1942) shows that a considerable swelling of the ranks of the younger criminals took place during the years 1915—1937. During this period crime was gradually becoming more

Table 2. **Boys 15—18 years of age convicted of serious offences during the years 1909—1914.**

Year	Boys: 15—18 years		
	Population 31/12	Convicted of serious offences	Per 10 000 in the corresponding agegroup
1909	157 834	84	5.32
1910	161 036	92	5.71
1911	161 566	57	3.53
1912	163 141	82	5.03
1913	163 866	96	5.86
1914	164 144	110	6.70

prevalent among the adolescents, a maximum being registered for the 15—18 year groups. This phenomenon is also discernible in DAHLBERG's (1943) investigation into the risk of being convicted of graver offences, referring to the year 1937. According to Dahlberg the odds are heaviest against young men between 15 and 18 and subsequently drop continuously. A woman in 1937 only ran a tenth of the risk incurred by a man, the odds being respectively 7.8 to 1 000 in women as against 72.4 in men. The figures quoted refer to the entire country. The figures for the towns differ, however, from those given for the country districts. The different risks in different age groups are presented graphically in Fig. 1.

From the survey by the Prison's Board the conditions during the period of 1921—1935 are illustrated in Fig. 2 by plotting the percentual deviations from the mean of the total apparent criminality as well as of the corresponding figures for young people between 15 and 18 during the different years. The greatest deviations from the mean in the juvenile subjects refer to −45 in 1922 and +50 in 1935, and between these two poles the graph runs a smooth course. A certain covariation between total and juvenile criminality is suggested by the diagram.

According to the survey quoted above, the female share in juvenile criminality has increased, amounting in 1935 to 12.6 per cent of the male.

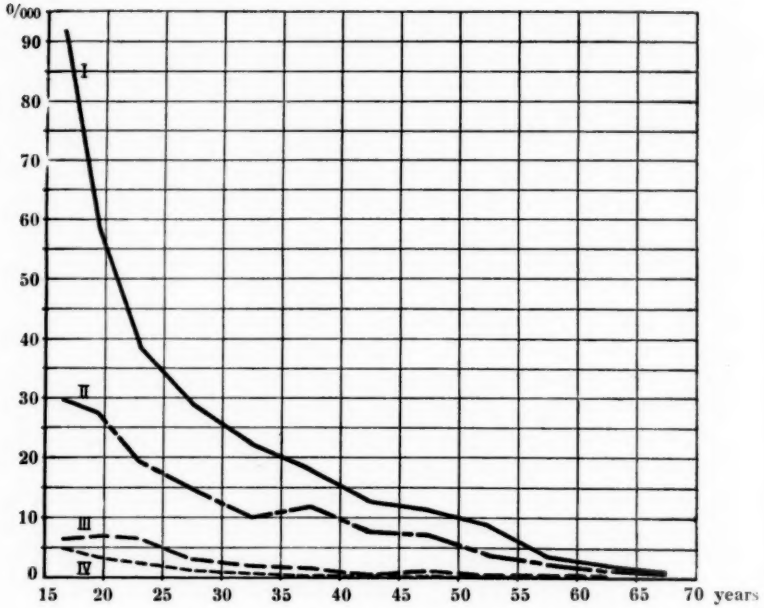


Fig. 1. The risk of being convicted of serious crime at different ages in the year of 1937 in Sweden. (after Dahlberg, 1943.)

Curves: I = men, towns, II = men, rural districts, III = women, towns, IV = women, rural districts.

AGE.

The age of the delinquents at the time of their being taken in charge is of comparatively slight interest. The term »delinquent» excludes adult individuals. On the other hand we can speak of delinquency only when the child has reached a certain age. The age is furthermore defined by the fact that protective institutions admit only children of a stipulated age (within narrow age limits). Existing reports on the age at admission are accordingly of slight interest only, but as they can presumably be used for the purpose of comparing different materials, a short review is given of available statements.

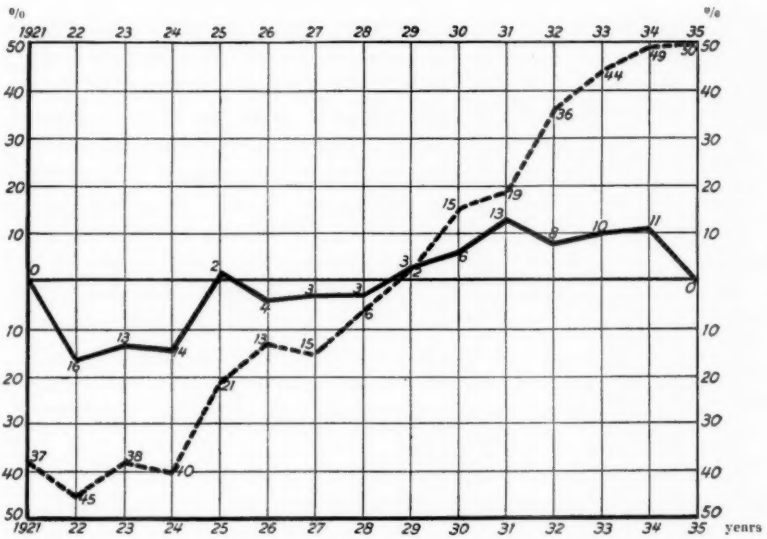


Fig. 2. Juvenile and total criminality during the period of 1921—1925. (after the survey by the Prison Board.) Cf. the text.

total criminality.

juvenile criminality.

HEALY and BRONNER (1926) were able to show an increase of numbers in each year group between 6 and 16 years, and subsequently a decreasing tendency, identical for boys and girls. The authors point out that their experiences consequently do not bear out the current opinion, according to which a steady increase until the age of 18—20 years is the rule. They consider the cause of this phenomenon has not been satisfactorily elucidated but suggest cooperation of different circumstances. The median age for boys in their paper lies between 14—15 years, whereas the greatest number of cases is to be found in the 13—14 years group. The median age for girls is between 15 and 16 years, the maximum numbers occurring one year earlier.

According to GLUECK and GLUECK (1934), the first symptoms of delinquency in a material consisting of boys appeared at a

mean age of 9.7 years, whereas the mean age at the first contact with the police was 11 years and 11 months, and the first appearance in the juvenile court took place at 13.5 years on an average. In an earlier work (1930) the same authors found that slightly more than 10 per cent had been in contact with the authorities even before the age of 10 years, 27 per cent before 14 years of age, 77.2 before 16 and 92.5 before 18 years. The average age of known first conflict was 14.8 years. The authors point out, however, that the true number of earlier contacts is presumably much greater, and consequently the average age is appreciably lower than that suggested above, owing to the impossibility of obtaining reliable information on this point. However, the last-mentioned material generally consists of older boys who had been sentenced to institutions corresponding to Swedish juvenile prisons.

Most of the German authors omit to tabulate the age distribution in their material merely giving a summary division into two groups »schulentlassene» (postschool age) and »schulpflichtige» (of school age) (GREGOR, GREGOR and VOIGTLÄNDER, MÖNKEMÖLLER, KLUGE). No account is given of the age of maximum occurrence.

GRUHLE (1912) however, points out that the thirteenth year is most critical for boys, a fact which is also evident in the work by Fuchs-Kamps.

ARCTANDER and DAHLSTRÖM (1932) review all age groups between under one and seventeen years of age; this is because their material contains not only delinquents but also children who have been taken charge of on account of unsatisfactory home conditions. Most boys as well as girls had been taken in charge at an age of between 11 and 15 years, however.

ARCTANDER (1936) in her later work has submitted her earlier material to a closer analysis. Accordingly the age distribution in the two papers is the same. She now offers the information that girls were admitted to »tvangsskole» (industrial school) at an average age of 8—15 years and to »skolehjem» (detention homes) of 9—18 years.

LUND (1918) maintains that the earliest signs of delinquency might appear as early as at 4—5 years of age, and it is not a

question of »normal» naughtiness only but of real delinquency, which subsequently becomes increasingly manifest. Symptoms of delinquency so early in life are exceptional, however. The author was further able to demonstrate that the graph representing the appearance of first signs of delinquency presented two maxima, at 9—10 years and 13—14 years respectively, separated by a minimum at 11—12 years. The average age of intervention by the board is 13.1 years for boys and 13.8 for girls, *i.e.*, no appreciable difference between the sexes.

AHNSJÖ (1941) gives an average age of 13.8 years for girls admitted to ordinary detention homes during the period of 1903—1925, while in a later series, covering the years 1926—1937, the corresponding age was found to be 15.0. As is pointed out by the author, the age depends to a certain extent on the legislation. Accordingly, the delinquents admitted to reformatories show a higher average age, 16.7 for both periods.

TYPES OF DELINQUENCY.

A definition of the term »delinquency» is not to be found in the comprehensive literature on its causes and prognosis. The need for a definition only arises when scientific analysis is in question — in practical work we can easily dispense with it. Most investigators have assumed delinquency to be an unambiguous concept requiring no explanation. A definition has to aim at practical and objectively recognisable features. If founded on psychic characteristics the definition becomes vague and indefinite and may be variously interpreted. Definitions which more or less clearly refer to causal factors are quite worthless. German authors who have discussed the problem (TÖBBEN, ZIEHEN, GRUHLE and others) without being able to agree on a conclusive definition, have been unanimous in their opinion that the perverse behaviour which we are accustomed to label delinquency originates in a predisposition of character expressing itself in asocial or criminal actions.

As the different types of delinquency have been very differently treated in the literature, it is necessary to discuss

the methods employed, before reviewing the investigations.

One would wish to divide the delinquents into categories according to such characteristics as, for instance, the indolent, the actives, the mendacious, the cruel, the unreliable and so on. Objective estimates of character are, however, impracticable. To test children with a view to probe their character is not likely to be successful. Instead it has been necessary to be content with judging by their behaviour and actions; the very act which has led to intervention by the authorities being as a rule, the point of departure.

In a comparative survey of the classification adopted by various authors a number of things have to be kept in mind in order to explain different conclusions. In the first place one must see whether a material is divided up according to criminal or asocial acts or to their perpetrators. Obviously the percentage in the first case is different because the same child might have committed a number of criminal as well as asocial acts. Where the latter principle of classification has been chosen the case might either have been labelled according to the nature of the most outrageous act registered, which as a rule means the infringement that has provoked the authorities, or else according to recurrent misconduct of a defined nature which lends its colour to the case. Definitions and classification in the works of earlier authors are fairly confused and the terminology far from uniform.

Another circumstance that makes it more difficult to judge of the frequency of different expressions of delinquency reported by various authors is the fact that some investigators have included heterogenous characteristics in the same categories. Not only are different degrees and kinds of delinquency indiscriminately lumped together but the terms themselves are applied alternately in a wider and a more restricted sense. The category »vagrancy», for instance, is hard to define and might include anything between playing truant from school and veritable tours of larceny. The group »general misbehaviour» frequently becomes a collective term for indefinite or insufficiently unravelled cases. The category sexual delinquency in its broadest sense might cover all degrees of sexual misbehaviour

from prostitution to promiscuity but might on the other hand in some instances only imply infringements of the Venereal Diseases Prevention Act or other sexual breach. Where girls are concerned, much depends on the extent to which sexual misbehaviour is judged and persecuted. In the case of girls such things are assumed to constitute a serious matter. Probably they are just as common among the boys but this is often looked upon as normal and in any case does not imply the same risks. Consequently the category sexual misdemeanour includes many girls.

Another circumstance to be borne in mind is that differences in the distribution of the groups of delinquency might be due to the source of the various materials. Naturally the origin of any material will colour its composition and has led to different results on such issues as the classification of crime and its causes or prognosis, and so on. Whether the cases go back some time or refer to recent years is also a point to be taken into consideration, because of its bearing not only on their frequencies, as mentioned before, but also on their quality. Where the headings are few they are given to the worst cases only but when many columns are available the slight cases are also recorded.

To a certain extent all classification of necessity becomes subjective and dependent on the manner in which the author has defined the limits of his group or settled the diagnosis in the particular case. A very small group in the report of one author might thus only reflect critical sifting and narrowly defined limits. S:SON FREY's (1944) examination of 600 girls from the clinic for psychic advice in Stockholm might serve as an illustration in this respect. The group of sexual delinquents — by him designated prostitutes — comprises only 40 cases or 6.6 per cent, whereas the category »general delinquency» numbered 377 cases or 62.8 per cent.

The distribution of different types of delinquency given by various investigators is shown in tables 3 and 4. The figures are, as has been pointed out before, not quite comparable but they might be taken to convey a comprehensive impression of conditions in general. Obviously offences against property

Table 3. **Types of delinquency in boys according to different authors.**

Author	Year	Country	Age	Number of cases	Types of delinquency											
					Theft [1]		Property offences of other kinds [2]		Vagabondage		Sex offences		General delinquency [3]		Combinations of all kinds	
					No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1. Lund	1918	Sweden	7—18	743	525	70.7	124	16.7	203	27.3	47	6.3 [4]	50	6.7		
2. »	1918	»	11—17	175	174	99.4	54	30.9	108	61.7	16	9.1	18	10.3		
3. Jonsson ...	1944	»	4—20	3352		63.4		4.6		2.4		3.7		18.9		7.0
4. Nissen	1917	Norway	11—19	300	245	81.7	10	3.3					45	15.0		
5. Arctander	1936	»	7—17	146	70	47.9			26	17.8	2	1.4	6	4.1	42	28.8
6. Skjerbæk ...	1929	Denmark	14—17	118	98	83.1			9	7.6	4	3.4	7	5.9		
7. »	1929	»	9—14	97	57	58.8	2	2.1	7	7.2	2	2.1	29	29.9		
8. Gruhle ...	1912	Germany	14—20	105		71.4		40.9		16.9		5.7		18.0		43.8
9. Gregor	1918	»	— [5]	54	48	88.9	32	59.3	42	77.8	5	9.3	35	64.8		
10. »	1918	»	— [6]	46	39	84.8	4	8.7	20	43.5	9	19.6	33	71.7		
11. Burt.....	1925	England	5—18	124		82.2		13.0		39.9		17.1		30.7		
12. Healy & Bronner	1926	U. S. A. Chicago I	6—17	694		75.5		3.2		85.0		8.5		21.0		
13. Healy & Bronner	1926	» » II	6—17	692		88.5		2.0		96.0		8.5		12.2		
14. Healy & Bronner	1926	» Boston I	6—18	758		97.3		1.2		98.0		9.0		20.8		
15. Healy & Bronner	1926	» » II	6—18	686		93.8		2.9		72.0		10.8		18.7		
16. Glueck & Glueck	1930	»	15—21	506	404	79.8	12	2.4	8	1.6	34	6.7	48	9.5		
17. Glueck & Glueck	1934	»	6—13	969	736	76.0	10	1.0	76	7.8	9	0.9	138	14.2		

[1] Including burglary.

[2] Including forgery or fraud, damage by fire, malicious damage.

[3] Including violence or cruelty to animals.

[4] One case of manslaughter.

[5] Children above school-age.

[6] Children at school-age.

Table 4. **Types of delinquency in girls according to different authors.**

Author	Year	Country	Age	Number of cases	Types of delinquency											
					Theft [1]		Property offences [2] of other kinds		Vagabondage		Sex offences		General delinquency [3]		Combinations of all kinds	
					No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1. Lund	1918	Sweden	7–18	106	68	64.1		1.9	7	6.6	2	1.9	1	0.9	46	43.4
2. Ahnsjö	1941	»	6—18	2005 [4]	462	23.0			130	6.5	645	32.2	335	16.7	433	21.6
3. »	1941	»	15–18	443 [5]	339	76.5			4	0.9	50	11.3	21	4.7	29	6.5
4. Jonsson	1944	»	4—20	1061		19.8		1.9		5.1		41.4		5.6		26.2
5. S:son Frey...	1944	»	15—20	505	88	17.4					40	7.9	377	74.6		
6. Nissen	1917	Norway	8—17	146	38	26.0							103	70.5	5	3.4
7. Arctander ...	1936	»	9—17	75	27 [6]	36.0			23	30.6	19	25.3	1	1.3	5	6.7
8. »	1936	»	—17	163	27 [6]	16.6	2	1.2	32	19.6	73	44.8	5	3.1	24	14.7
9. Skjerbæk ...	1929	Denmark	16—18	43	25	58.1			2	4.7	14	32.6	2	4.7		
10. » ...	1929	»	11—14	18	10	55.6			1	5.6	3	16.7	4	22 2		
11. Gregor &	1918	Germany	— [7]	71	27	38.0	12	16.9	41	57.7	43	60.6	44	62.0		
12. Voigtländer	1918	»	— [8]	26	21	80.7	5	19.2	6	23.1	4	15.4	20	76.9		
13. Burt	1925	England	7—18	74		43.3		1.4		19.8		37.9		35.1		
14.	1926	U. S. A. Chicago I	8—17	306		32.0		3.0		42.5		63.0		35.3		
15. Healy &	1926	» » II	8—17	308		30 0		1.5		51.0		74.5		26.0		
16. Bronner	1926	» Boston I	8—17	242		40.0		0.8		81.6		53.2		16.5		
17.	1926	» » II	8—17	314		41.0		0.3		72.3		55.8		16.4		

[1] Including burglary.
[2] Including forgery or fraud, damage by fire, malicious damage.
[3] Including violence or cruelty to animals.
[4] From detention homes.
[5] From reformatories.
[6] Including forgery and fraud.
[7] Children above school-age.
[8] Children at school-age.

constitute the most frequent indictment against boys in Sweden as well as in other countries and during different periods.

HEALY and BRONNER (1926) made a study of 4 000 juvenile delinquents in two American towns, Boston and Chicago. Their material was made up of young boys and girls who in 1909—1914 were brought before the juvenile courts of these towns. In order to eliminate minor offences such as selling without license, peddling and the like only recurring cases were included.

The authors found that between 80—90 per cent of the boys had participated in stealing covering petty larceny or illicit »car borrowing» and house-breaking. Fraud and forgery were extremely rare, less than 3 per cent. A certain difference was noticeable between the two towns of Chicago and Boston, crimes of violence and robbery being substantially more common among the delinquents from the former. If vagrancy, truancy and other kinds of migratory tendencies are added up, the resulting group becomes almost larger as that representing property crimes and acquisitiveness. Infringements such as excessive mendacity or conspicuous laziness which we would have labelled general delinquency amount respectively to 14 and 10 per cent. Sexual misbehaviour was relatively rare among the boys — only 8—10 per cent — while such misconduct dominated among the girls in all series, the figures being 53 and 56 per cent for Boston against 63 and 75 per cent among the girls from Chicago.

In conformity with previous authors, GLUECK and GLUECK (1934) were able to register the majority of the offences or 76 per cent as property crimes. Truancy and running away was noticed in 7.8 per cent only. Crimes of violence (2.4 per cent) were as rare here as with previous authors. Sexual misdemeanour (0.9 per cent) was relatively seldom found as was to be expected in an investigation dealing with boys alone.

The difference discernible between certain groups — among others, those concerning vagrancy — in the respective works of these authors, whose materials come from the same country, may presumably be due to the fact that Glueck and Glueck have only recorded the principal indictment in each case,

whereas Healy and Bronner have included all the misdemeanours of which the youngsters have been guilty.

BURT (1925) puts crimes of acquisitiveness committed by boys at 91.9 and by girls at 46 per cent. Sexual misconduct reaches 37.9 per cent in girls but also shows relatively high figures (17.1 per cent) among the boys. Vagrancy is more common among boys than among girls but general delinquency and in particular mendacity is more often met with among the girls.

GRUHLE (1912) in his material of boys naturally finds a high percentage of property crimes, i.e. 77.1, but vagrancy only represents 17, and sexual misconduct 6 per cent.

GREGOR (1918) distinguishes between symptoms of delinquency and criminal actions. Amongst the latter, theft constitutes the largest category with 89 per cent. Damage, mischief and the like were met with in 18.5 per cent. 13 and 33 per cent were sentenced for begging and vagrancy, respectively. If common tramps are included amazingly high figures (almost 80 per cent) are reached. It is, however, not evident how the figures have been arrived at. Obviously, nearly all the boys have been exhibiting several symptoms of delinquency and been guilty of various criminal acts as well. These figures refer exclusively to the group labelled »schulentlassene» (post-school) by the author, which only numbers 54 cases and accordingly is far too small to admit of general conclusions. The author shows a similar distribution in a second group of 46 »schulpflichtige» (boys of school age). The property crimes dominate enormously, general delinquency coming second. Damage, mischief and so on curiously enough figure less often, although the group is younger.

GREGOR and VOIGTLÄNDER (1918) were able to register about 60 per cent of sexual misconduct among post-school girls (schulentlassene), general delinquency and vagrancy coming next, followed by larceny. In the younger group of girls attending school (schulpflichtige) property crimes dominate with 80 per cent and general delinquency appears in 77 per cent.

ARCTANDER (1936) records 47.9 per cent of property crimes and 17.8 per cent of vagrancy among the group of boys labelled

»representative selection». Simultaneous instances of more than one type of misdemeanour amounted to 28.8 per cent. Among the younger girls property crimes account for the highest figures, vagrancy holds the second place, while sexual misconduct is met with only in slightly more than one-fourth of the cases. The group of elder girls displays the usual distribution with sexual misconduct taking precedence, being recorded among half the total number. Signs of general delinquency occurred in both categories but only in few instances. The disparity shown can be accounted for by the difference in age.

LUND (1918) also shows the greatest number of boys taken in charge because of property crimes recorded in 70.7 per cent; next in frequency comes vagrancy at 27.3 per cent, followed by other kinds of misdemeanour such as mischief, damage and outrage to morality, all of them represented by low percentages. The serious offences which have landed their perpetrators in a detention home for severe cases consist, according to the author, to 99.4 and 61.7 per cent of theft and vagabondage respectively. Among the girls offences of theft also rank foremost (64.1 per cent), a combination of theft and sexual misdemeanour coming next with 43.4 per cent, while isolated sexual offences only reach 1.9 per cent. Vagrancy is also fairly rare, 6.6 per cent only.

In the Swedish Report, *Gang criminality,* (Ligabrottsligheten), 1941, the crimes of greed also predominate. Against 432 gangs specializing in theft, house-breaking and similar activities stand only 8 and 13 gangs devoted to fraud and embezzlement respectively. Other types of crime were not represented. Strictly speaking, fraud and embezzlement should be included among the property crimes, which accordingly constitute the purpose of criminal associations.

AHNSJÖ (1941) whose work was carried out at the State Institute of Human Genetics in Sweden has conducted a most thorough analysis of a large material composed entirely of women and introduces the nomenclature »type of delinquency». He divides his girls into different groups. In the first place come the four elementary groups, sexual misconduct, property crimes, vagrancy and general delinquency. Four additional groups refer

to cases displaying a combination of different types of delinquency. The author is able to show that the group denominated sexual misconduct account for the highest frequency, or 32.2 per cent, followed by property crimes at 23 per cent and general delinquency in 16.7 per cent of the cases. An analysis of the correlation between age and types of delinquency demonstrates that acquisitiveness either by itself or combined with vagrancy or general delinquency is more common in the younger age groups of 6—11 years than is the case in the group over 15 years, where sexual misconduct singly or in combination with vagrancy reaches comparatively high frequencies.

As far as I know no corresponding investigation into the correlation between age and types of delinquency has been made among boys. Most authors, on the other hand, have focussed their attention on the appearance of the first sign of delinquency and the manner in which it manifests itself. As has been pointed out previously, American authors (HEALY and BRONNER, OWENS, GLUECK and GLUECK) consider truancy to reveal delinquency at an early age.

ROBISON (1936) was not able to substantiate this assertion, finding on the contrary that the truants over 10 years were 13 times as numerous as those below that age. German authors are also inclined to look upon truancy as an early symptom of delinquency. GRUHLE (1912) thus quotes 64.7 per cent of his cases as making their début as truants or vagabonds.

LUND (1918) who found the first signs to be theft in 48 per cent and vagrancy in 45 per cent points out that these two kinds of misdemeanour are frequently found together, so that it is impossible to form an opinion as to which of them first becomes manifest. It is presumably safe to maintain that all statements concerning either the age at which the first signs of delinquency present themselves or the nature of these latter must be considered as fairly doubtful.

Because of the difficulty in fixing any definite age at which delinquency begins, it is also hard to tell to which type of delinquency the first symptoms belong. When a child grows up and starts behaving badly, its naughtiness is at first frequently considered to be charming and evidence of precosity.

Later we start talking about bad habits and of a spoilt child. Only when the child becomes still older do we speak of delinquency. Parents as a rule show more patience than others but obviously the age at which the authorities deem intervention necessary also varies.

CAUSES OF DELINQUENCY.

Just as treatment of diseases becomes less successful if not founded on knowledge of aetiology and pathology, so do all endeavours to treat the problems presented by criminality and delinquency become unsatisfactory where insight is lacking in the deeper underlying causative factors. Accordingly efforts have been made along different avenues of approach to form an opinion as to why men turn criminal, and children develop the particular variety of misbehaviour which we call delinquency. The answer to the question has been looked for mainly in two domains: the predestinate rôle played by heredity, and the inevitable influence of environment on adjustment to social life. Opinion has changed in the course of time as to which of these factors should be accorded the greatest importance, from the time when Lombroso introduced his conception »the born criminal» right up to the prevailing American saving faith in education of our days.

When weighing heredity against environment the significance of such social conditions as are common to all citizens can be ruled out. With reference to environment, one must consider economic and other differences between the classes which cause certain groups to be worse off and faced by greater difficulties in life, but the narrower environment represented by home conditions also has to be taken into account.

It goes without saying that home conditions and the qualities of the parents constitute the environmental factors exercising the earliest and most intense influence on the children, in particular during their sensitive and receptive infancy. Great importance has accordingly been attached to home and growth conditions in such investigations as the present. Different

anomalies have been regarded as causative factors in the origin and development of delinquency, acting either by themselves or in connection with other circumstances. In discussing the importance of the home, the reservation must be made from the start that although the home represents the most intimate environment of the delinquents, its quality might depend on the ability of the parents to create a good atmosphere and accordingly also be conditioned by heredity. In other words, certain hereditary qualities in the parents might either be transmitted directly or influence the offspring in a roundabout way via a constitutional inability in the parents to provide proper surroundings for their children. A certain parallelism between the qualities of parents and children respectively and the character of the children and the atmosphere in the homes can be expected, and the causal connection might in both instances be determined by heredity. Accordingly the theoretical interpretation of the obtained results is not unambiguous. If these reservations are kept in mind, however, an examination of home conditions is undoubtedly of great interest.

One of the conditions of special interest is that of the children growing up in incomplete families either due to illegitimacy or to what is technically known as »broken homes»; these cases ought to be examined separately, although they are often included under the heading »bad homes».

ILLEGITIMACY.

The question of the extent to which the delinquents were born out of wedlock ranks high, particularly in Swedish investigations, and great importance is ascribed to illegitimacy as a causative factor in the development of delinquency.

The percentages of illegitimate births have been comparatively high in Sweden during the later part of the nineteenth and, particularly, during the twentieth century, the figure referring to 1930 being 16 per cent for the whole country. Since then a gradual decrease has set in. In 1943 a rate of illegitimacy of 8.6 per cent was registered — the lowest figure so far. When comparing the Swedish figures with those reported from other

Table 5. **Frequency of illegitimate births in different countries according to official statistics.**

Country	Year	Per cent illegitimate of the total number of births	
		Towns	Rural districts
1. U. S. A.	1935	10.3	
2. England	1937	4.2	
3. Germany	1900	8.7	
4. »	1910	9.0	
5. »	1923	10.4	
6. Portugal	1941	15.1	
7. Sweden	1891—00	10.9	
8. »	1901—10	20.6	10.3
9. »	1911—20	22.0	12.6
10. »	1921—25	19.8	13.1
11. »	1926—30	20.4	14.3
12. »	1931—35	17.2	14.5
13. »	1936—40	13.3	12.3
14. Norway	1898—1900	7.6	
15. »	1940	6.4	
16. Denmark	1941	8.6	
17. »	1943	8.9	
18. Finland	1939	7.7	
»	1942	6.9	
19. Island	1941	22.0	

countries during the latest decades, our figures appear comparatively high and are exceeded only by those registered in Iceland (22 per cent) and Portugal (15 per cent). Our Scandinavian neighbours report figures between 6 and 8 per cent, Germany 8—10 per cent and England 4 per cent. Illegitimate children were formerly more common in the towns than in the country, but of late an adjustment has been noticeable whereby the towns at present only register slightly higher rates than the rural areas. Whereas the percentage of births out of wedlock during the eighteenforties was 7 per cent in the country and 27 per cent in the towns the conditions have

Table 6. **Frequency of illegitimate births among delinquent boys and girls according to different authors.**

Author	Year	Country	Total number of cases	Illegitimate births		Character of the cases
				No.	per cent	
Boys						
1. Lund.....	1918	Sweden	743	170	22.9	
2. »	1918	»	175	47	26.9	severe cases
3. Fritzell ...	1935	»	822	219	26.6	
4. Jonsson...	1944	»	3 352	—	14.6	Born in towns
5. » ...	1944	»		—	16.3	» » rural districts
6. Arctander	1936	Norway	345	30	8.7	Juvenile crim. cases [1]
7. »	1936	»	232	39	16.8	Random cases
8. Skjerbæk	1929	Denmark	97	27	27.8	Detention homes [2]
9. »	1929	»	118	30	25.4	» » [3]
10. Gruhle ...	1912	Germany	105	14	13.3	
11. Burt	1925	England	123	—	6.5	
12. Healy &	1926	Chicago U.S.A.	2 000	—	2.6-2.8	Boys + girls
13. Bronner	1926	Boston »	2 000	—	2.0-2.8	» » »
Girls						
1. Lund.....	1918	Sweden	106	31	29.2	
2. Fritzell ...	1935	»	345	115	33.3	
3. Ahnsjö ...	1941	»	931	289	31.0	Born in towns
4. » ...	1941	»	813	216	26.6	» » rural districts
5. Jonsson...	1944	»	1 061	—	21.6	» » towns
6. »	1944	»		—	25.5	» » rural districts
7. Arctander	1936	Norway	110	14	12.7	Industrial school (tvangskole)
8. »	1936	»	180	29	16.1	Detention homes
9. Skjerbæk	1929	Denmark	43	6	14.0	» » [2]
10. »	1929	»	18	8	44.4	» » [3]
11. Barth.....	1915	Germany	40	7	17.5	
12. Burt	1915	England	74	—	9.5	

[1] »Vergerådsgutter».
[2] For older cases (ungdomshjem).
[3] For younger cases (skolehjem).

changed to such an extent that the corresponding figures for 1935 were 13.9 and 15.1 per cent respectively.

The table containing extracts from a few representative investigations in this field illustrates the variations between different countries. Undue importance obviously cannot be attached to a comparative survey of this kind, because general frequencies differ from one country to another. Moreover, in some instances the material is so small as to create chance variations.

American authors attach little significance to illegitimacy. HEALY and BRONNER (1926) quote the figures 2.0—2.8 per cent which appear remarkably low.

BURT (1925) in his material registers 7.6 per cent as born out of wedlock contrasted with 0.7 per cent in the control group. A difference of this magnitude suggests nonconformity in his controls which have presumably been taken from socially better classes. A comparison of the figures with figures from England (3—4 per cent) indicates twice as many illegitimates in the official statistics, whereas by contrast the control figures appear to be abnormally low. The rate of illegitimate births presumably being higher in London than in the rest of the country, it is hard to form an opinion as to the real state of things.

Among German authors GRUHLE (1912) in particular has shown interest in this question. By comparison with the official figures and taking into account the higher mortality among children born out of wedlock, the author arrives at the conclusion that illegitimate birth was 3—5 times as frequent among the delinquents as among the rest of the male youths. In his own material Gruhle numbers 14 illegitimate boys (13.3 per cent). The mothers of these boys were not found to be particularly criminal, addicted to alcohol or mentally abnormal; nor did an examination of the boys reveal conspicuous inferiority. The author maintains and substantiates by still more arguments his assertion that unhappy conditions during childhood should be responsible for tendencies towards delinquency to a far greater extent than hereditary disposition. The material, however, is far too small to admit of any conclusion whatever.

From Norway only ARCTANDER (1936) goes into this question. In a group labelled criminal boys (kriminella vergerådsgutter) the author registers 8.7 per cent illegitimately born, whereas 16.8 per cent of the boys in a group called representative selection were born out of wedlock. The figures in the two girl groups, those from industrial schools (»tvangsskole») and the ones from detention homes (skolehjem) were 13 and 16 per cent respectively. In the three last-named categories the rate of illegitimacy was accordingly twice as great as that in the total population, whereas the first-mentioned group, representing graver offenders, only shows figures slightly in excess of the general standard. The figures for the town of Oslo are presumably higher than those referring to the rest of the country, but the author offers no information on this point.

SKJERBÆK (1929) in Denmark reports that 57 out of 215 boys (26.5 per cent) were born out of wedlock. Although taken from a small material this figure agrees fairly well with Swedish findings.

LUND (1918), for instance, found 22.9 per cent of the boys in ordinary detention homes and 26.9 per cent of the boys in a severe detention home to be born out of wedlock, against 11.4 per cent of illegitimacy in his control group, the calculated differences being respectively 11.5 ± 1.0 and 15.5 ± 3.6 per cent. The corresponding figures referring to girls were 29.2 against 10.4 per cent, or a difference of 18.8 ± 5.3 per cent.

When dividing up his material from a detention home for severe cases in children with hereditary disposition, and environmentally conditioned children, the author registers a relatively high percentage of illegitimacy (31.7) in the former group as contrasted with 23.5 per cent in the latter, and postulates a causal connection between delinquency and illegitimacy. In his opinion the children who were born out of wedlock owe their criminality to bad hereditary disposition, even if they grow up in depressing surroundings. The difference between the two categories, i.e. »anlagsbarn» and »miljöbarn» was found to be 8.2 ± 8.3 per cent. The division of the children according to heredity and environment obviously rests on the subjective ground of the author's personal acquaintance with the children.

From the scientific point of view the method of division must accordingly be considered worthless. It is, in addition, evident from the calculated mean error that the material is too small to warrant any conclusions.

We must also take into account the fact that, at the time from which Lund's material originates, the social ostracism was severe and might have been instrumental in increasing the difficulties of life of the illegitimates who presumably also had to be taken in charge more often than other children.

AHNSJÖ (1941) also shows the rate of girls born out of wedlock in his material to be greater than among the total population. The relatively high proportion of illegitimates among the delinquents is explained by the author to be due to neglected education because the mothers who have to earn their living are not able to devote sufficient time to their children. In addition the unmarried mother has fewer possibilities of taking care of a child who shows a tendency to delinquency and is less able to prevent interference by the authorities. Furthermore, the mental make-up of the unmarried mothers intellectually as well as in other respects is in the author's opinion inferior, and these defects are transmitted to the offspring, in which they become manifest as delinquency.

AHNSJÖ on further analysis of his material from detention homes is able to show that the frequency of detained girls is higher among country girls born out of wedlock than among the same girls coming from the towns. According to the author the high urban illegitimacy is only apparent. It is well known that parents in the towns frequently live together without being married — conduct which is seldom tolerated in the country. Their children accordingly enjoy all the advantages of home life and are less liable to decline into delinquency.

AHNSJÖ was able to make a most interesting observation. During the war years 1914—1918 the rate of illegitimacy among the detained delinquents, from towns as well as country dropped below the average, a phenomenon not observed during any other period. Why children born in wedlock during years of crisis will run a greater risk of being detained as delinquents, is in the authors opinion difficult to explain. He supposes that

in periods of crisis the absence of the father on military service and the necessity for the mother to earn her living outside the home partly eliminate the difference between the conditions of the legitimate and the illegitimate child. The matter might also be explained in the following way. Delinquency became more widespread during these years of war, making it necessary to pay more attention to the quality and seriousness of committed infringements than to home conditions when deciding on protective measures.

There are, however, also investigations in which the frequency of illegitimates does not exceed that current among the rest of the population. In both of the investigations carried out by The Central Union for Social Work, Criminality gangs (Ligabrottsligheten 1941), and Juvenile criminality (Ungdomsbrottsligheten 1941) the percentages of illegitimacy are not particularly high, rather the contrary. In the investigation first mentioned, where the material originates from the big towns of Sweden, the rate of illegitimacy during the years 1930—1939 was found to be 15.9 per cent, thus showing no difference from the official figure (16.3 per cent) referring to the same period. In the last-mentioned report on juvenile delinquency the figures tabulated in the various groups were found to differ. The highest rate of illegitimacy, i.e. 25 per cent, was recorded under the heading »frauds, embezzlers etc.» while the category »assaulters» only showed 10 per cent of illegitimate boys. For one thing these figures, however, refer to older groups; further, they are derived from smaller towns and rural districts treated as a unit, and are accordingly hardly comparable with previous investigations. The statistics issued by the Central Prison board 1942 for the years 1915—1937 again yielded a different result, recording relatively many delinquents as born out of wedlock.

Jonsson (1944) in his report concerning the psychic care of children and adolescents also points out that illegitimacy is more common among delinquent children from the country than in a corresponding clientèle from the towns. Furthermore, the percentage of children born out of wedlock is higher among the girls than among the boys.

BROKEN HOMES.

The term »Broken Homes» is of common use in most social investigations, and implies the breaking up of the home through divorce or in consequence of the absconding or death of one of the parents. Children from such homes obviously have to face harder conditions when growing up than their happier comrades from homes kept together by both parents. Great importance has accordingly been ascribed to these circumstances which have been considered as instrumental in leading young people astray. In investigations from abroad, 'broken homes' figure more conspicuously than is the case in our surveys.

HEALY and BRONNER (1926) found broken homes in just under 45 per cent of their huge material composed of 4 000 young persons from the two great towns of Chicago and Boston. The same report contains information regarding 675 children from Chicago, among whom the rate of broken homes conformed with the figure quoted above, 49 and 53 per cent for respectively boys and girls. The death of parents was one and a half time as frequent a cause of dissolution of homes as separation or divorce.

GLUECK and GLUECK (1930) reports 60 per cent of broken homes, of which two thirds were due to death of parents; divorce or absconding being responsible for the rest. The latter group includes such instances where homes have become defective due to lengthy detention of one of the parents in an asylum, hospital or prison. The authors also include homes where the supervision of the children has been continuously inadequate, whereby the total of 'broken homes' amounts to 84 per cent. In a later paper by the same authors (1934) the figures quoted are not quite as high. 'Broken homes' in its narrower sense now accounts for 45.5 per cent. No controls are mentioned, but the authors refer to another investigation into a number of Chicago families, according to which a little less than one seventh of the families had been scattered because of the death of one parent or through divorce.

BURT (1925), reporting on a London material, shows broken

homes to be twice as frequent among juvenile delinquents as in a control group, and points out that the girls in this respect are more unfavourably placed than the boys. In all 60 per cent of the criminal children came from homes in which family life was defective in one way or other.

German authors do not use the nomenclature 'broken homes', but have for a long time focussed their attention on the deterioration of environment suffered by children who lose both or one of their parents.

SPANN (1905) was one of the first to study this problem, and was able to register a substantially greater number of orphans among the delinquents. Among conscripts in Frankfurt the same author found 7.21 per cent of old offenders among the men both of whose parents were living. The corresponding figure for those who had lost father, mother, or both parents were 9.94 per cent, 11.5 and 10.28 per cent respectively. Gruhle, from whom these figures have been quoted, remarks that more observations are needed to make sure whether orphans actually always have a better social outlook than those who have lost their mothers only, or if the phenomenon is a peculiarity in the material analyzed by Spann.

MÖNKEMÖLLER (1911) found 33 per cent of orphans in his material and in addition 19 per cent whose parents had been separated — in all, 52 per cent of dissolved families. Considering that a further 20 per cent were born out of wedlock his material was certainly heavily loaded from the point of view of unhappy upbringing.

GRUHLE (1912) reports on about 8 per cent of orphans. Slightly more than 25 per cent had lost their fathers and just under 24 per cent their mothers. The author does not state to what extent children of divorced parents are represented among his cases.

GREGOR (1918) attaches very little importance to environmental factors such as upbringing, considering the endogenous causes to be decisive. Only in 14 cases are »zerrüttete Familienverhältnisse» (rotten home conditions) held responsible. In a few cases the conditions during the first world war were blamed.

ARCTANDER (1936) quotes instances where the father has either died or left the home. In her two groups of boys this has happened in 19.1 and 18.5 per cent respectively (66 out of 345 and 43 out of 232). The corresponding figures relating to her girl groups were 30 and 31.7 per cent respectively (33 out of 110 and 57 out of 180). The loss of the father accordingly constituted a much more ominous handicap for the girls. The difference amounted to 12.6 ± 4.1 per cent and was statistically significant.

LUND (1918) does not use the term 'broken homes' which was coined at a later date, but he has investigated the conditions covered by the term. In doing so he was able to register 21.8 per cent (185 out of 849) who had lost their fathers, as contrasted to 15 per cent (122 out of 815) in his group of controls, a difference of 6.8 ± 1.9 per cent. 16.5 per cent (140 out of 849) had lost their mothers against 9.1 per cent (74 out of 815) in the control group, the difference being 7.4 ± 1.6 per cent. Delinquents had accordingly lost one parent more frequently than children belonging to the group of controls. The value of the controls, however, is questionable, as will be discussed later. Loss of the father occurs more frequently than loss of the mother. This is least obvious with regard to the girls but most conspicuous where the boys from the »severe» detention home are concerned. The difference among the latter was 7.4 ± 4.2 per cent. It appears natural, however, that the loss of the father should have most effect in such cases, because such a loss means a lowering of the financial standard of the family. The mother is compelled to take on outside work, and supervision slackens. Where the splitting up of the homes is due to the absconding of one of the parents, the difference in comparison with the controls becomes still more manifest 13.1 per cent (84 out of 641) as against 4 per cent (29 out of 718) or 9.1 ± 1.5 per cent. Death of one parent was more often the cause of the breaking up of a home than separation. Only in such cases where the father died or left the home can a significant difference between the effect on boys and girls respectively be traced; the girls being most adversely affected, even more so than the boys from the detention home for severe cases, the

figure being 11.7 per cent (9 out of 77) against 9 per cent (51 out of 564) among the boys admitted to ordinary protective homes. The author, however, does not consider himself entitled to draw any conclusions from his findings as to the significance of the discussed circumstances for the development of delinquency.

AHNSJÖ (1941) has registered home conditions with regard to supervision of girls. Whereas 67.9 per cent (1 663 out of 2 448) were at birth living with both parents, only slightly more than 50 per cent remained with both parents up to the time when they were taken in charge. The parents were divorced or separated in 10.8 per cent, and in 32.7 per cent the homes were broken up by death. The father had died in 12.3, the mother in 17.5 and both parents in 2.9 per cent. Accordingly, a girl who had lost her mother runs a greater risk of becoming delinquent than a girl losing her father, the difference being 5.2 ± 1.2 per cent. Thus, almost half the number of homes (43.5 per cent) were broken at the time when the girls had to be taken in charge. Comparing the clientele from reformatories with that relegated to detention homes, no difference could be found, whether the parents were married or only living together, nor in these cases where the parents were dead. By contrast divorced parents appear at a statistically significant greater frequency among the pupils in detention homes than among those sent to reformatories, the difference being 4.4 ± 1.4 per cent. The frequency was found to be greater during the later period (1926—1937) than during the earlier (1903—1925). The difference between the two periods worked out at 6.3 ± 1.5 per cent. Children whose parents are divorced apparently run a greater risk than other children of being taken in charge on account of delinquency. This, according to the author, can hardly be due only to the fact that parents are missing, because if such were the case, orphans and children who have lost one parent through death ought to be as often taken in charge, all the more so because of their greater number. Obviously other circumstances are at work. The author considers that heredity may possibly play a part, and is inclined to look upon divorce as imputable to indifferent hereditary qualities which reveal

themselves as delinquency when transmitted to the children. The author may be right in this, but there are no proofs of the assertion that illegitimate children are the bearers of hereditary qualities inferior to those possessed by children born in wedlock.

From the Gang Criminality (Ligabrottsligheten 1941), originates the statement that 30 per cent of the gang members hail from broken homes, divorce being accountable to a slightly greater degree than death. The high percentage by itself tells us very little about the causes of delinquency, because it is natural for parentless children to join gangs. In any case, conditions differed in the different towns. In Malmö, for instance, more than every fifth of the examined boys came from homes dissolved by divorce, while the figures from Stockholm and, in particular, Gothenburg are lower.

In Juvenile Criminality (Ungdomsbrottsligheten 1942), a grouping has been made according to the nature of the crime, such as perpetrators of violence, thieves and others. When investigating the immediate background of the crimes the different factors, although showing general conformity, appear to be of different importance in the different groups. On account of death or divorce the families were broken in 22 to 28 per cent in the different groups of the material.

JONSSON (1944) reports that 63.6 per cent of the children had been brought up by parents living together. The boys seemed to be better off in this respect than the girls, 68.2 as against 49.3 per cent respectively living with both parents. 'Broken homes' were met with in one fourth of his cases, this factor being more ominous for girls than boys. If legal divorce and other kinds of separation are lumped together, the figure representing 'divorce children' amounts to 10.5 per cent. According to the author this indicates the children whose parents have been divorced to be 2—3 times as numerous among the delinquents as among children in general.

REGNÉR (1943) found not less than half the number of children to originate from 'broken homes'. His material, however, is very small and was specially selected.

Thus, investigations so far reported indicate 'broken homes', which term has been variously defined, to appear more often

in the records of the delinquent than among the population in general. The inquiries are not extensive and only few authors (Lund, Burt) have supplied controls, but the figures arrived at by them ought to be considered as conclusive, all the same. Other authors have also arrived at high figures, although lacking control material. When estimating such results great care must be exercised. Under otherwise identical conditions, a defective home naturally has to be considered as a drawback implying a lowering of the environmental standard, but many children who grow up in such families show normal development and become useful citizens. This environmental factor can hardly be decisive by itself, but might be looked upon as a contributary cause. In addition the inadequate faculty of collaboration and lack of balance sometimes instrumental in bringing about divorce might occasionally reflect inheritable psychic defects. In such cases environment need not be determinative, the leading part being instead played by heredity. Where homes have been broken up by the death of one of the parents, we are dealing with quite a different matter. On this account the concept 'broken homes', employed in American investigations, is not very satisfactory. Homes disrupted because of death ought to be kept statistically apart from homes which have become defective because the parents have separated on account of dissension and the like. In the available literature no division along these lines has been made, presumably because a more correct and thorough analysis of the causative factors would necessitate a splitting up of the material in too small groups from a statistical point of view.

In the works of earlier authors circumstances such as poverty, alcoholism and criminality play an important part in the records of delinquent juveniles; an exact estimation, however, of their import has not been made. Strictly speaking, figures admitting of comparison ought to be available. As GRUHLE (1912) rightly points out our conclusions regarding the problem of delinquency would rest on safer foundations if we knew the number of non-criminal children whose fathers are drunkards, mothers criminals, or parents infirm, and also the

rate of bodily infirmity, psychic abnormalties or peculiarities of temperament among such children.

The only authors employing such controls are BURT and LUND.

BURT's (1925) delinquency clientèle is made up of 123 boys and 74 girls chosen among cases referred to him privately or in his capacity of director of an office for the examination of juvenile delinquents. Accordingly, it is a question of selected cases, of which the author is in possession of full particulars. The controls consist of 400 non-delinquents of the same age who belonged to the same social class, went to the same school and were living in the same district.

Only 8 per cent of Burt's controls came from poor homes. This figure indicates the controls to belong to a socially better class than the delinquents, precluding any comparison.

LUND (1918) uses as controls an almost equal number of wellbehaved children, only the 'good' boys being slightly fewer. As need hardly be pointed out, the finding of a material suitable for the purpose of comparison from the point of view of social standing and other characteristics is attended by great difficulties, and certain objections may also be raised to this control material. The author, who has collected his controls partly from orphan asylums, partly from elementary schools situated not only in the suburbs of Stockholm and Gothenburg but also in smaller towns and in the country, considers these children as belonging to the same social strata as the delinquents. Obviously, however, considerable discrepancy exists concerning, for instance, the economic standing of the different families. It may admit of doubt whether the author's estimate of the adequacy of his controls is warrantable. When he interprets demonstrable differences such as, for instance, the greater poverty in the families of the delinquents as one of the reasons why children fail to adapt themselves, he certainly seems to be rushing to conclusions. Such disagreement only indicates dissimilarity between the two compared categories. 61.3 per cent of the delinquent girls had furthermore grown up in the big towns which, however, only furnished 41.5 per cent of the controls, a circumstance which cannot fail to be of importance.

ECONOMIC FACTORS.

In foreign investigations great stress has been laid on poverty as a cause of crime and delinquency. Poverty is obviously not only more pronounced, but its consequences are also more sinister in countries where the social conditions are less satisfactory than in Sweden. Even if it can be shown that parents of delinquents are on an average poorer than others, little significance may be attributed to such a finding. Poverty might operate by creating a general deterioration of environment, but it is also possible that parents have become paupers because they have certain inheritable qualities causing a general ineffectiveness. The results of this kind of investigations are accordingly not unambiguous. An estimation as to whether poverty is making its influence felt directly or indirectly is hard to arrive at in investigations of this type. Other obvious factors of equal importance might easily escape recognition. It is also hard to decide whether or not poverty is present, as this conception, like others, may be defined in various ways.

HEALY and BRONNER (1926) divided their 675 cases into 5 economic groups. The lowest category, the destitute, including families deprived of the barest necessities of life, only numbered 5 per cent. The opposite extremity, represented by families living in luxury, included about the same number, or 4 per cent. This of course was to be expected, since wealthy parents of this class are better able to make private arrangements for their delinquent children, and thus prevent interference by public authorities. As poor, were designated families living from hand to mouth, amounting to 22 per cent. 35 per cent were registered as 'normal', represented by people with little more than adequate means of subsistence. The category 'comfortable', making 34 per cent, drew an income permitting of ordinary pleasures and of providing pocket-money for the children. It is amazing that only 5 per cent belonged to the destitute. A far greater quota from this group could reasonably be anticipated among the delinquents. Analyzing their huge material of 4 000 individuals the same authors were able only to register poverty in 8 and 24 per cent respectively in the two Chicago groups, as

compared with 19 and 16.7 per cent in corresponding groups from Boston. However, where there is no information about the make-up of the total population from the point of view of social classes, no conclusions can be drawn regarding the origin of the delinquents from this or that class.

GLUECK and GLUECK (1934), reporting on 925 cases, classified 219 or 23.7 per cent as 'comfortable', 631 or 68.2 per cent as belonging to the 'marginal group' and the remaining 75 or 8,1 per cent as 'dependent'. The last-mentioned group was made up of persons permanently on the dole, while those who were just able to make a living were accounted for in the marginal group. The purport of the figures, however, is hard to judge, because controls are lacking.

BURT (1925), dealing with a London clientele, includes in the category 'poor and very poor' the children whose parents were only casually employed. 19 per cent of the delinquents came from such families, as compared with 8 per cent of the average London population. The latter figure appears to be strikingly low. The group labelled 'small but regular income' contained 37 per cent of the cases, and corresponded to 22 per cent of the total population. The largest group, 42 per cent, is made up of relatively well-to-do parents such as artisans, small shopkeepers and the like. The remaining upper-middle-class and aristocracy only contribute 3 per cent of delinquents, although making 18 per cent of the population. In discussing his findings the author, pointing to the 56 per cent brought up in poor homes, is inclined to consider it as proved that poverty constitutes one of the incitements to crime; but penury can hardly be the only or even the most important factor, because after all not less than 46 per cent of the delinquents were born of relatively well-to-do parents.

Most German authors do not analyse the importance of poverty as a cause of delinquency, the problem being merely touched upon in passing.

MÖNKEMÖLLER (1911) found that 36.8 per cent of his 623 cases had 'very poor' parents.

GREGOR (1918) considered poverty to be one of the environmental causes of delinquency only in 5 cases out of 95.

LUND (1918) divided his cases into 4 groups, according to the financial position of the parents. After having excluded the homeless children, one-third were found to originate from financially sound homes and two-thirds from poor or very poor families. The boys from the detention home for severe cases and the girls were worst off. In the control groups, the conditions were reversed. As has been pointed out, the conclusion arrived at by the author, according to which poverty is looked upon as a causative factor in the development of delinquency and crime, appears to rest on a very precarious foundation.

ALCOHOLISM.

Alcoholism is quite as subjective a conception as poverty, and is judged of differently in different countries and provinces as well as in different social classes. Consequently, clear-cut and objective statements of a nature to enable comparison are hard to get.

HEALY and BRONNER (1926) do not distinguish between abuse of alcohol, immoral life and criminality in the homes, which are all included in one group. In their large series of 4 000 cases such conditions could be shown to obtain in 21 per cent, an unexpectedly low figure considering that the material is derived from Chicago.

BURT's (1925) investigation deals with conditions in London during recent years. 12.2 per cent of the boys and 16.2 of the girls had fathers described as drunkards, contrasted with 1.5 and 3 per cent respectively in the control material. If inebriety at home was considered only when occurring in close temporal connection with the taking care of the children, the figures were only 6.5 and 10.8 per cent respectively or on an average 8.1 per cent. The author, however, points out that abuse of alcohol is very common in the social surrounding of these children and appears three times as often in the homes of the delinquents as among the controls. The great difference, however, might be due to less thorough inquiries about the homes of the control children.

From GRUHLE's (1912) survey referring to many countries, it appears as if the parents of delinquents as well as juvenile criminals admitted to reformatories were frequently considered to be alcoholics. The figure quoted for England is 29 per cent. Fluctuations of between 23 and 50 per cent were found in different states of the U.S.A. Chicago showed 72.2 per cent. From Germany figures ranging between 11 and 30 per cent are given, adult criminals reaching even higher. The import of these figures, however, is hard to estimate, because for one thing they refer to earlier times, when the consumption of alcohol was more widespread, for another they have been compiled from different materials which were presumably judged differently. Another drawback is that controls for comparison are lacking. In his own material of juvenile delinquents GRUHLE was able to prove abuse of alcohol by one or the other of the parents in 35 per cent, a figure considered by him as a minimum.

Of greater interest to us are similar investigations from our Scandinavian neighbours. These, however, are very few; to my knowledge, only one from Norway being available.

ARCTANDER (1936) uses two different designations for misuse of alcohol. Thus 'alcoholism', either isolated or combined with other qualities such as criminality, was found among the parents of the two groups of boys criminal boys (kriminella vergerådsgutter) and 'representativt urval' (representative selection) in 61.3 (168 out of 274) and 67.3 (113 out of 168) per cent respectively. The corresponding findings concerning girls from industrial schools (tvangsskole) and detention homes ('skolehjem') were 51 (43 out of 84) and 40 (48 out of 121) per cent respectively. In addition the author employs the term »drikk hos faren eller moren» (father or mother addicted to drink) a condition which appears in the records of the girls in 49 and 30.5 per cent respectively. In summing up, the abuse of alcohol can be said to be met with remarkably often in the homes of delinquents.

LUND (1918) conducted his investigations at a time when the temperance boards had not yet started their activities. No authority is quoted for his facts, which probably originate

Table 7. **Frequency of different characteristics in parents of delinquent boys.**

Author	Year	Country	No. of fathers and mothers with known characters [1]	Criminality		Alcoholism		Loose habits		Intellectual defects		Mental abnormity	
				No.	%	No.	%	No.	%	No.	%	No.	%
Fathers													
Lund	1918	Sweden	564 (743)	71	12.6	206	36.5	—	—	28	5.0	31	5.5
»	1918	»	149 (175)	21	14.1	59	39.6	—	—	13	8.7	11	7.4
Arctander	1936	Norway	274 (345)	62	22.6	168	61.3	8	2.9	—	—	13	4.7
»	1936	»	168 (232)	16	9.5	113	67.3	9	5.4	—	—	6	3.6
Gruhle	1912	Germany	67 (105)	47	70.1	34	50.7	—	—	—	—	14	20.9
Gregor	1918	»	65 (100)	16	24.6	25	38.5	2	3.1	—	—	9	13.8
Burt	1925	England	100 (126)	—	5.7	—	12.2	—	3.3	—	6.5	—	6.5
Mothers													
Lund	1918	Sweden	645 (743)	17	2.6	45	7.0	112	17.4	43	6.7	53	8.2
»	1918	»	170 (175)	11	6.5	13	7.6	42	24.7	10	5.9	14	8.2
Arctander	1936	Norway	274 (345)	—	—	—	—	8	2.9	—	—	—	—
»	1936	»	168 (232)	—	—	—	—	9	5.4	—	—	—	—
Gruhle	1912	Germany	83 (105)	35	42.2	6	7.2	—	—	—	—	12	14.5
Gregor	1918	»	41 (100)	8	19.5	4	9.8	9	22.0	—	—	1	2.4
Burt	1925	England	100 (126)	—	0.8	—	7.3	—	13.8	—	13.8	—	8.1

[1] No. of children in the whole investigation in brackets.

Table 8. **Frequency of different characteristics of parents in delinquent girls.**

Author	Year	Country	No. of fathers and mothers with known characters	Criminality		Alcoholism		Loose habits		Intellectual defects		Mental abnormity	
				No.	%	No.	%	No.	%	No.	%	No.	%
Fathers													
Lund	1918	Sweden	77 (106)	3	3.9	25	32.5	—	—	3	3.9	2	2.6
Ahnsjö	1941	»	293 (2448)	26	8.9	18	6.1	—	—	16	5.5	170	58.0
Arctander	1936	Norway	84 (110)	9	10.7	43	51.2	16	19.0	—	—	6	7.1
»	1936	»	121 (180)	20	16.5	48	39.7	24	19.8	—	—	9	7.4
Gregor & Voigtländer	1918	Germany	53 (100)	14	26.4	16	30.2	2	3.7	—	—	9	17.0
Burt	1925	England	74	—	2.7	—	16.2	—	6.8	—	4.3	—	2.8
Mothers													
Lund	1918	Sweden	85 (106)	3	3.5	2	2.4	10	11.8	5	5.9	3	3.5
Ahnsjö	1941	»	288 (2448)			20	6.9			14	4.9	225	78.1
Gregor & Voigtländer	1918	Germany	51 (100)	14	27.5	1	2.0	8	15.7	—	—	6	11.8
Burt	1925	England	74	—	4.1	—	20.3	—	25.7	—	8.2	—	6.8

[1] No. of children in the whole investigation in brackets.

from the extracts of records kept by the Child Welfare Board which accompany the children to the detention homes. The statements to be found in these records accordingly reflect the opinions of different examiners, and must be considered as more or less subjectively coloured. Lund shows 36 per cent of his delinquent clintele to have drunkards as fathers. Among the boys admitted to a severe detention home the rate was still higher, i.e. 40 per cent, as contrasted with 8.1 per cent in the control group, a difference of 27.9 ± 2.15 per cent. A marked difference between the two groups was also found in such cases where the mothers were the victims of alcohol: for the whole material 6.4 against 0.4 per cent, with a difference of 6.0 ± 0.93 per cent. The difference was greater among the boys than the girls.

JONSSON (1944) gives the figures 15.6 and 0.7 per cent for the rate at which the father or mother respectively are quoted as being »given to drinking». The homes were afflicted with abuse of alcohol in 15.8 per cent. These figures are considerably lower than those given by LUND. It is hard to say whether this discrepancy reflects a true general change during the 26 years which have elapsed between the two investigations, or if it indicates incomplete information about the Jonsson material, an explanation which the author himself seems inclined to accept. JONSSON also owes his facts to the Child Welfare Board, and it would appear strange indeed if the conception 'abuse of alcohol' had been less rigorously applied during later years. Presumably, however, the term alcoholism has been differently defined by LUND and JONSSON.

CRIMINALITY OF THE PARENTS.

Obviously the moral standing of the parents makes itself felt in all walks of life, but it becomes particularly conspicuous when reflected in criminal actions. Previous authors have arrived at very different results which to a certain extent might be explained partly by the different interpretation given to the conception 'criminality', partly by the sources from which the information has been supplied.

American authors do not pay great attention to this question.

HEALY and BRONNER (1926), as mentioned before, quote only the total percentage of criminality, including alcoholism and immorality.

GLUECK and GLUECK (1930) submit 51.5 per cent criminal homes (207 out of 402 families). They, however, do not confine themselves to counting the parents only, but include instances where criminal acts were perpetrated by brothers and sisters or near relatives, as well as inebriety. The figures only refer to infringements committed before the time when the boys were taken in charge. In a later work (1934) the same authors also quote high figures, 57.9 per cent of issued sentences. When including such infringements of the law as had escaped the notice of the authorities, 26.3 per cent were added. The authors arrived at a sum total of 86.7 per cent in which some relative of the juvenile delinquent had been convicted of crime.

BURT (1925), reporting from London, quotes very low figures for criminality, his averages being 4.6 and 2 per cent for father and mother respectively (boys and girls together). 'Vicious conduct' by either of the parents appears in 6.5 per cent among the boys and 8.1 per cent among the girls as compared to 0.5 and 0.2 per cent respectively in the control material.

German investigations report comparatively high criminality figures, ranging from 27.7 (BARTH) to 47.7 per cent (MÖNKEMÖLLER).

GRUHLE (1912) registers higher figures still, one or both of the parents in his material having served a sentence in 71.8 per cent. The author considers the high percentage to be due to thorough inquiry. The circumstance that vagabonds and fined persons were included might, however, also serve as an explanation. The point is discussed whether these children came from 'criminal families', a term defined as families in which several members are professional criminals. 7.6 per cent (8 out of 105) of the boys admitted to the Flehinger reformatory were brought up in 'criminal families' as thus defined.

Only ARCTANDER (1936) reports figures from Norway. No percentages are given, only net figures for the different groups.

When working out the percentages it is necessary to start either *only* from those persons of whom information is available or, as has been done by the author in other respects, from the *whole* material, the different proceedings yielding slightly different results. Using the first mentioned method the percentages in the two groups of boys work out at 22.6 and 9.5 per cent respectively (62 out of 274 and 16 out of 168) — one or both parents — the corresponding figures for the two groups of girls being 10.7 and 16.5 per cent respectively (9 out of 84 and 20 out of 121).

LUND (1918) did not find parental criminality to the same extent in his Swedish material as was the case in the Norwegian investigation. His information was extracted from the parish registers in the places where the parents had been living for a long time. One or both of the parents were found to have served a sentence in 11 per cent (86 out of 783) of the whole material and 17.1 per cent of the boys from a severe detention home (29 out of 170) as contrasted with 1.0 per cent (8 out of 791) of the controls, the difference being 10.0 ± 1.2 and 16.1 ± 2.9 per cent respectively. The fathers are naturally represented by higher figures than the mothers. The mothers of the worst cases, however, showed a criminality rate of no less than 6.5 per cent, a finding worthy of consideration, especially when compared with the incidence of criminality among the mothers of boys in common detention homes, worked out to be 2.6 per cent, or less than one half of the first-mentioned rate. Lund did not include vagabonds sentenced to forced labour or fined persons. Considering that Lund collected his facts at a time when the parish registers were fairly regularly notified, his compilation is of considerable interest, particularly because he has taken the trouble to check his files by perusing the 'Police Gazette'.

IMMORALITY IN THE PARENTS.

Obviously, the moral standard of the parents may be wanting in other respects than those dealt with above. Not to be overlooked are bad sexual habits becoming manifest as lascivious-

ness and promiscuity. As pointed out by several authors the overcrowding frequently met with in poor families in earlier days, accentuated by the custom of taking in lodgers, causes sexual acts to be staged before raised curtain. The children become witnesses to more or less improper conduct by their parents or by strangers. Sexual immorality has always been looked upon with greater severity when displayed by women than is the case where men are concerned, presumably because such conduct make the women more conspicuous and entails greater risks for them. In consequence the investigators have attached greater importance to the behaviour of the mothers than to that of the fathers in this respect.

BURT (1925) lumps together sexual immorality, drunkenness, nagging, lack of supervision and so forth under the heading 'vicious homes' which figure in 51.5 per cent of his cases, to be compared with 9.7 per cent among the control group. He points out that parents very seldom directly encourage their children to commit crimes or walk the streets.

ARCTANDER (1936) does not register specific figures concerning sexual misconduct, employing instead the sweeping heading: 'other defects of character, immorality and so on in one or both of the parents'. The great difference in this respect between boys and girls, each sex being subdivided in two groups, constitutes a feature of particular interest in her investigation by showing the girls to be more seriously menaced. The actual figures for boys were 2.9 (8 out of 274) and 5.4 (9 out of 168) per cent respectively in the two categories, corresponding to 19.0 (16 out of 84) and 19.8 (24 out of 121) for the girls. The differences amount to 16.1 ± 4.4 per cent between the lowest and 14.4 ± 4 per cent between the highest figures.

LUND (1918) examines the mothers from the point of view of lascivity; 112 out of 645 or 17.4 per cent of the boys and 11.8 per cent of the girls (10 out of 85) were found to be born of promiscuous mothers, the figure regarding the boys from Hall (the severe detention home) attaining 24.7 per cent (42 out of 170). In the control group of boys (21 out of 653)

the corresponding figure was 3.2 per cent, whereas no case was recorded among the girl controls.

In contrast to Arctander's findings the difference between boys and girls is not great, and the boys appear to be worse off than the girls. Leaving out the boys who come from the detention home for severe cases and constitute a selection of particularly bad cases, the difference amounts to 5.6 ± 3.8 per cent and is accordingly uncertain.

In addition to the parental shortcomings discussed so far, i.e. qualities favouring the development of alcoholism, criminality and lasciviousness, other psyshic characteristics have been investigated which, although primarily of genetic importance, must also be considered to influence the environment. The qualities in view refer to the mental equipment of the parents intellectually as well as in the field of emotions and will power, anomalies coming under the heading of psychopathy. The upsetting of the balance in these two psychic dominions is disclosed in many ways, but is considered to exercise a particular influence on the discipline and supervision of the children. Other external circumstances, such as where mothers are compelled to work for a living away from home, obviously react on the bringing up of the children and ought accordingly to be taken into account. Some authors have stressed these deficiencies without, however, always distinguishing between external and internal causes.

HEALY and BRONNER (1926) register insufficient supervision in 23 and 46 per cent respectively in the two series from Chicago and 51 and 39.5 per cent respectively in the Boston series. The authors also attach great importance to discord between the parents as indicative of their mental equipment and report c:a 14 per cent of such homes.

BURT (1925) groups together cases where discipline has been slack on account of moral or intellectual instability, and records 32 per cent of such homes among the delinquents against 3.2 per cent in the controls. Insufficient supervision due to other causes figures at 21.9 per cent against 3.9 in the control groups.

In conclusion, previous investigations can be said to have shown that in many respects the homes were of poor quality

and frequently displayed serious deficiencies. How often the parents have been good and the homes above reproach is, however, a matter of conjecture.

HEALY and BRONNER (1926) have to a certain extent given an answer to this question. Having ruled out all the homes commented upon in any way, the remaining good homes were found represented at a rate of 10.3 and 5 per cent among 1 000 cases of the Boston and Chicago material respectively. Among 2 000 young offenders who have relapsed the homes were to be considered as fairly good, only in 7.6 per cent.

ARCTANDER (1936) designates the parents positively as 'good' in 27.7 (76 out of 274) and 25 (42 out of 168) per cent of the boys as compared with 20.2 (17 out of 84) and 24.8 (30 out of 121) per cent among the girls. On an average the parents were pronounced to be satisfactory in one-fourth of the cases. This of course cannot be construed as implying 25 per cent of the homes to be good because, owing to different circumstances, the parents might be prevented from exercising necessary control over the children.

ENVIRONMENTAL CONDITIONS OUTSIDE THE HOME.

Several authors have approached the question concerning the importance of comrades, and been inclined to look upon 'bad company' as one of the causes not only of criminality but of general maladjustment as well. To obtain unequivocal facts of a nature to sustain or disprove such opinions, however, is not easy in investigations of this type and the results reported by different authors are contradictory.

HEALY and BRONNER, (1926) analyzing 3 000 cases, consider 'bad companions' to be the cause of delinquency in 62 per cent. The rate was practically the same in the Chicago and Boston investigations, as well as in both sexes. The latter is noticeable as girls seldom are members of gangs.

BURT (1925) is willing to impute a certain, although subordinate significance to fellowship. The conflicting results arrived at by the quoted authors are hard to explain. The calculations were presumably carried out in different ways, and

probably refer to different conditions. HEALY and BRONNER who report high figures are likely to have examined the participants in criminal acts. Practical experience goes to show that enterprises such as theft, housebreaking and the like are seldom undertaken single-handed, but are usually the work of gangs which are said to abound in Chicago and Boston, a circumstance calculated to account for the high estimates in their investigation. BURT's low figures, on the other hand, might imply that only such cases have been included where either a direct incitement to criminal or asocial conduct could be proved or in cases of delinquency where a contagious influence was discernible.

Only few authors discuss the formation of juvenile gangs under the aspect of a sprouting social evil, which it is imperative for society to nip in the bud.

TRASHER (1927) has made an interesting study of gangs among juveniles in Chicago. He points out that the gangs often begin as innocent unions and far from always develop a threatening attitude towards society. Doubtless, however, a substantial proportion of the juvenile league members swell the ranks of gangsters.

GRUHLE (1912) draws attention to the danger inherent in such leaguing together, and quotes in support several authors (STARKE, LIEPMAN, HERZ, LINDHEIM, POLLAK). Among his own boys only 11.4 per cent belonged to gangs.

In the above quoted work »Ligabrottsligheten published» by the Central Union of Social Workers a gang was defined as a union for criminal purposes of not less than 3 persons who had been guilty of at least 3 crimes. During the years 1938—1940, 322 gangs of this category numbering 1 595 members were active in the three largest towns, Stockholm, Gothenburg and Malmö. It is hardly necessary to point out that gangs in Sweden never attain the same prevalence, cohesion or menacing character as in America, being as a rule formed by small boys. Most juvenile gangs confine themselves to the rifling of slot machines and the breaking of kiosks. As a rule there are no ringleaders, and when caught the members try to fasten the blame on each other. Gangs of this kind are soon found out,

and are hardly a public danger. A noticeable increase in the number of gangs has been registered during the last few years.

Just as gangs are to be found only in towns, the contingent of delinquents is mainly recruited from the towns, a circumstance, according to GRUHLE, which is commented on by several authors (SCHOTT, THOMA, BAER).

GRUHLE (1912), however, points out that absolute figures are of little significance and stresses the necessity of comparing such figures with the number of young people of the same age in the corresponding districts. In analyzing his material, the author refers to the locality where the delinquents grew up rather than to the place where measures were taken, the latter possibly being quite fortuitous. Employing this principle, the author is able to prove that well over half of his clientele came from towns with more than 20 000 inhabitants. This cannot be interpreted offhand as branding the youth of the towns as inferior to those from the country. It must be kept in mind that the temptations are greater and the struggle for employment more intense in the great towns, where at the same time police supervision is more effective than in the country. In addition, considerations of an economic nature might restrain rural authorities from resorting to detention.

NISSEN (1917) was led to expect comparatively substantial levies from Oslo owing to the peculiar conditions in this town. So far from being the case, however, the contribution from the 14—15 years groups turned out to be smaller than predictions founded on the town's share in total criminality would suggest. The capital holds its own in this respect even where the 16—17 years old are concerned, but when boys older than 18—19 years are considered the proportion is reversed, Oslo making a comparatively greater contribution to these year groups than to the total criminality. The author is inclined to give the Child Welfare Board all the credit for the favourable figures in the age groups under 18 years. The good result presumably depends on the fact that delinquent youngsters are not registered as criminals when taken in charge by the Welfare Board, and accordingly it appears to be only a question of labelling.

LUND (1918) was able to show that 73.2 per cent of the worst

cases — the boys from the severe detention home — came from the towns, the remaining 26.8 per cent originating from purely rural districts which, however, at that time (1914) contributed more than 75 per cent of the total population. In the remainder of the author's clientele, consisting of 743 boys and 106 girls, the difference between town and country was still more pronounced, inasmuch as 83.9 per cent came from urban communities and only 16.1 per cent were brought up in the country.

AHNSJÖ (1941), investigating the birth places of the girls, found the lowest returns from sparsely populated areas, and an increase in frequency corresponding to the density of settlement. When with the domicile at the time when measures were taken as starting point, the shift towards more thickly populated districts was still more marked than was the case when working on the birth-place quota. Concurring in the conclusions arrived at by other authors, Ahnsjö considers the difference as probably due to the increased temptations in the towns, and above all to their more effective administration reflected in prompter dealing with delinquents.

In the same way as the towns have been shown to contribute a greater contingent of delinquents, certain districts particularly in the great towns, were found to produce more delinquents and thus warrant the denomination 'delinquency areas', a matter well illustrated by TRASHER (1927) in his investigation of the gangs in Chicago.

ROBISON (1936) has surveyed the American investigations on this subject. The most important of these, carried out by CLIFFORD R. SHAW, abounds in remarkable and interesting observations. In the first place, pronounced conformity was shown regarding the frequencies of truants, delinquent youngsters and criminals in the different districts of Chicago. The rate was found to vary in inverse ratio to the distances from the centre of the town, in such a way that the percentages of truancy, delinquency and crime became higher as the centre of the town was approached. Furthermore, a marked interrelation of the three phenomena could be proved, a high rate of delinquent youths being shown by increased percentages of truancy as well as of crime. A certain relation to the housing standard

in the different areas could also be traced, districts of poor houses registering greater percentual proportions of truancy, delinquency and criminality.

Similar investigations in other American towns have yielded corresponding results.

BURT (1925) collected information regarding the districts in which those children were domiciled who were sentenced to reformatory education in London during the years 1922—1923. It turned out that the cases were fairly evenly distributed over the inner and older parts of the metropolis with the exception, of the City proper. The author states that a substantial number of his cases were living in the slum districts, where theft and housebreaking constitute the principal means of living for their fathers.

CRÉMIEUX, SCHACHTEL & COTTE (1945) have conducted a similar investigation regarding the living quarters of 3 000 delinquents in Marseilles during the years 1933—1944. The greatest concentration occurred in the dock areas, apparently the oldest part of the town, from where a fanwise centrifugal decrease in the frequency of delinquency was noticeable.

QUALITIES IN THE PARENTS.

Although various kinds of environmental influence must be considered as of great importance and very often presumably play a decisive part in the development of delinquency all such cases cannot be accounted for in this way. The significance of genetic factors also have to be considered.

However, it is very difficult to isolate these factors and most authors have contented themselves with giving information about shortcomings in the parents of different kind. Especially one has tried to find out the frequency of psychopathy among them.

HEALY and BRONNER (1926) do not use the expression psychopathia but register the occurrence of 'abnormal mentality' in 7 per cent in both series of their cases.

CRÉMIEUX, SCHACHTEL et COTTE (1945) only found mental aberrations in 3.98 per cent of the parents.

GRUHLE (1912), in 23 out of 105 cases, i.e. 21.9 ± 4.0 per cent, considered at least one of the parents to be suffering from »geistig Abnormitet». The author, however, does not make clear the scope of this expression.

LUND (1918) devotes himself to this problem in a comprehensive investigation. The mothers throughout were shown to be of a lower mental standard than the fathers, 16.7 ± 1.4 per cent of the total material having mentally defective mothers (122 out of 730), as against 73 out of 641 or 11.4 ± 1.3 per cent of defective fathers. The difference is 5.3 ± 1.9 per cent and is not significant but probable. In some cases both parents were mentally deficient. The total amounts to 21.1 per cent (165 out of 783), a figure in fairly good agreement with that reported by GRUHLE. The girls were not more represented than the boys. The highest figures recorded 27.6 ± 3.4 per cent (47 out of 170) refer to the boys from the severe detention home.

The author ends his survey by pointing out that more than one fifth of all the children had parents of whom one at least was mentally defective, while the corresponding rate among the controls was one twentieth. The objection can, of course, be raised that different conditions have been included by the author under the heading mentally defective ('psykiskt defekta'). Thus, feeble-mindedness, insanity, epilepsy, and hysteria were all lumped together. The actual frequency of psychopathy proper does not, follow from these figures.

When analyzing sexual irregularities, the conduct of the mothers, as said before, has been considered to be of paramount importance as an environmental factor in the development of delinquency in girls. Some authors, however, maintain that loose living in mothers might be due to a passionate disposition which could be transmitted to the children and, in the present connection, in particular to the girls. The sexual misdemeanour constituting the principle type of delinquency in girls would then be of genetic origin. No proof of the correctness of this assumption can be found in the available literature, only few authors contributing elucidatory figures.

BURT (1925) estimates sexual misconduct by either of the parents to appear in 22.8 per cent of the records of delinquents as against 5.2 in the control group. Closer scrutiny reveals the girls to be more heavily burdened (25.7 per cent), while the boys remain at a lower level (13.8 per cent).

Because objective dividing lines are lacking, it is uncertain whether the controls have been judged in the same way as the delinquents, a circumstance which necessarily diminishes the value of the investigation.

LUND (1918) reports lewdness, »Liederlichkeit», in 16.7 per cent of the boys' mothers (122 out of 730) contrasted with 2.8 per cent among the controls (21 out of 755).

Examinations of twins ought to yield valuable results when extended to delinquents. Only few investigations of this kind have been carried out, however, and the material is obviously limited. The research in this field has mostly been focussed on adult criminals.

LANGE's (1928) material consists of thirty pair of twins. Thirteen of these pairs were monozygotic. Of these, both twins had been convicted in ten instances. The proportion was reversed among the dizygotic twins, where both twins had only been sentenced twice, while among the remaining 15 pairs of twins only one of them was punished. The discordant result concerning the three pairs of monozygotic twins is partly explained away by the author, who regards at least two of the cases to be the sequel of external causes — head injuries.

LEGRAS (1932), who has also examined criminal twins, found among them 4 monozygotic pairs in complete concordance, whereas only one of the twins in each of the five dizygotic pairs was involved (quoted from ROSANOFF). The number of cases is very small and the result might be due to chance, an objection which can also be applied to the material described by Lange.

ROSANOFF, HANDY & ROSANOFF (1936) have a larger material which they divide into three groups, designated respectively as adult criminals, delinquents and problem children. The first group consisted of 97 pairs of twins, of which 37 pairs were of the same sex and presumably monozygotic. Out of the 33 male pairs both twins were convicted in 22 instances, and one

of the twins only in 11 instances. Both twins of the four female pairs had been convicted in 3 instances. Of the remaining female pair only one was sentenced. Reverse conditions prevailed among the dizygotic twins, among whom both were involved twice, and one only in each of the remaining 20 pairs. The group designated delinquent twins shows a still greater concordance between the monozygotic twins. The authors, however, do not feel entitled to draw any conclusions from this other than that similar conduct can be expected where heredity and environment are like. The power of hereditary disposition is, however, exemplified by the authors in their account of two twin sisters who were separated at the age of three years and brought up in different localities. When sixteen both of them revealed sexual tendencies, started walking the streets and so on, and later both were admitted to detention homes at the same age. The dizygotic twins, however, as a rule also kept step with each other on the downward path. In the authors' opinion endogenous factors are insignificant as compared with postnatal influence in the creation of delinquency, an assertion which holds good for boys as well as for girls. Very little information is given about the third group, the problem children.

To a certain extent comparable with the twin researches reviewed above would be the investigation of groups of siblings who share more or less the same genetic disposition, and in consequence can be expected to show fewer hereditary variations than is the case with non-related persons. ARCTANDER (1936) has paid close attention to the sisters and brothers of the children taken in charge, and followed up 1521 out of 1955 children from 389 families. The majority, 78.5 per cent had been taken in charge by the Child Welfare Board on account of bad home conditions or because of delinquency. Among the others 102, or 6.7 per cent, had been reprimanded by the Board while only 225 children or 14.8 per cent did not appear in the files, and accordingly might be used as controls. As the author also considers bad home conditions which ought to apply to all the siblings alike, her results are hard to assess.

GRUHLE (1912) and LUND (1918) have attempted a practical

solution of the problem of the importance of heredity and environment respectively in the development of delinquency. Both authors have personally examined the individual cases and studied their records meticulously. The materials have subsequently been divided into groups according to the chief causes of delinquency. The three groups were children with hereditary disposition ('anlagsbarn'), children subject to environmental influence ('miljöbarn') and children in whom both hereditary and environmental influences could be shown ('såväl anlagssom miljöbarn'). In the two first-mentioned groups hereditary disposition and environment respectively have been considered the root of the trouble, whereas both factors were held responsible in the third group. The question concerning the causes of delinquency can hardly be expected to be answered in this way. When delinquency in a particular case is attributed to environment only, a purely subjective estimation is expressed which might be correct or wrong, but in any case is not proved. Without special and thorough investigations, no conclusions regarding this difficult question can be arrived at.

Opposite extremes of opinion regarding the importance of disposition and environment respectively in the development of delinquency are held by German and American authors. The Germans stress the endogenous factors which they are inclined to consider as the main cause.

GREGOR (1918) assigns first place to hereditary disposition, and quotes in support of his view the high figures found in his investigation. Among the two groups of boys 'of school age' and 'past school' 46 and 54 cases respectively, i.e. 95.4 and 86.5 per cent, were genetically handicapped i.e. by having abnormities in the family which were assumed to be hereditary. Only in 4 cases had no information been obtained. The hereditary disposition transmitted directly from the parents was paramount, and in more than half of the cases both parents were the bearers simultaneously. Siblings came next in frequency, whereas other relatives were few (only 11 cases). Among thus handicapping factors were counted mental and nervous diseases, psychopathy, suicide, alcoholism, criminality, lewdness and abnormal character.

GREGOR & VOIGTLÄNDER (1918) report on almost analogous conditions among 26 girls of school age and 74 girls who had left school, the actual figures being respectively 92 and 86.6 per cent hereditarily tainted. The factors are the same as those enumerated for the boys, alcoholism and criminality weighing heaviest. The material at the disposal of these two authors, however, is so small as to make conclusions precarious.

HEALY & BRONNER (1925), representing the totally opposite opinion, look upon hereditary disposition as a supplementary finding without bearing on the final issue. An examination of 675 juvenile delinquents revealed no instance of alcoholism, criminality or abnormal mentality among any of the parents or grand-parents in 316 cases, or 47 per cent. Among the remainder, alcoholism was found in 32, criminality in 24 and abnormal mentality in 17 per cent. In a considerable number of cases both the parents had these characters. The authors think the significance of these figures to be enhanced when viewed in the light of prognosis. The distribution of 'success' and 'failure' respectively did not seem to favour those in whom a genetic disposition could be ruled out, the failures being evenly distributed among all the groups at a rate of slightly more than 50 per cent. The authors consider themselves to be entitled to generalize when they maintain that the chances in favour of a happy development are even whether or not a hereditary disposition can be traced.

QUALITIES IN THE CHILDREN.

The investigations so far reviewed, however, refer not only to such qualities in the parents which could be assumed to cause maladjustment when transmitted to the children, but also to the presence in the children themselves of qualities which were of old considered hereditary. According to a widespread belief, irregular conduct of various kinds, criminal as well as asocial, is linked with the intellectual level of the parties concerned. As early as the 18th century the French philosopher Jean de la Bruyères stated: 'Si la pauvreté est la mère des crimes, le défaut d'esprit en est le père'. This conception also

rests on the provision in the criminal law according to which an individual must be in full command of his senses in order to be responsible for his actions. Incidentally, it may be mentioned that children and insane persons were placed on the same footing in the criminal codes of olden times because both were considered to be deprived of their full senses. Where the aetiology of crime is concerned great importance has been attached to the intellectual level. Intelligence has been looked upon mainly as a preventive or restraining factor, although it cannot be denied that a good intellect might be an asset in the world of crime also. Where adult criminals are concerned, certain categories of crime have been shown to be related to criminals of different intellectual level. Thus, swindlers and embezzlers are highest on the intellectual scale, while incendiaries and sexual criminals rank lowest, in fact seldom exceed the level represented by the lower mental classes. A similar relationship might exist between various types of delinquency, but so far as I know, only Burt has made an analysis of this kind which will be gone into later.

For practical purposes, the conception »intelligence» has for a long time been graded: the lowest stages — idiocy, imbecility and feeble-mindedness — to be followed by normal intelligence, gifted, talent and brilliance. Between the categories feeble-minded and normal BRANDER (1936) inserts a group called 'plain stupidity', corresponding to the 'physiological stupidity' referred to by other authors. The lowest grades are often covered by the heading 'defective intelligence', by psychiatrists termed oligofrenia.

Table 9 shows the different nomenclature employed by different investigators, and demonstrates the vagueness of the border-lines between different grades of intellectual defects.

Even before the introduction of intelligence test series, the different groups of oligophrenics were for practical purposes divided according to age. As »idiots» were labelled individuals whose intelligence did not exceed that of a normal three-year-old. Imbeciles corresponded intellectually to the 3- to 7-year-old, while the feeble-minded attained a degree of mental development corresponding to 7 to 12 years. The introduction by Binet-

Table 9. **Survey of the limits drawn by different authors in determining intellectual degrees. (According to Dahlberg 1937)**

Intelligence Quotient	Mental age of an adult	Terman	Wimmer	Burt	Bobertag	Brander	Swedish committee on mental deficiency, 1936	Swedish commitee on sterilisation, 1936
	15							
90	14	Dullness, rarely classifiable as feeble-mindedness	Inferioritas intellectualis Danish: Sinker			Physiologische Dummheit		
80	13				Fraglich debil			
	12	Border-line deficiency, sometimes classifiable as dullness often as feeble-mindedness		Retarded	Debil	Grenzfälle zum Schwachsinn	Mental class children (= Debiles)	//////////
70	11		Oligophrenia danish: Aandesvaghed Debile					
	10	Definite feeble-mindedness Morons			Imbezill	Debilität		Debilitas
60	9							
	8						Mental deficiency Feeble-mindedness Susceptible to instruction (= Imbeciles)	//////////
50	7		Imbecile					
40	6	Imbeciles		Subnormal Feeble-minded				Imbecillitas
	5					Imbezillität		
30	4						Non-susceptible to instruction (= Idiots)	//////////
20	3	//////////	Idioter					
10		Idiots				Idiotie		Idiotia
0	0							

Simon of series of tests for the grading of intellectual standard signified a great advance. This method, which was originally designed for the purpose of weeding out backward children from the normal school classes, had been worked out from examinations of a great number of normally gifted children. In the course of time the method has been modified and extended by several workers, e.g. BOBERTAG in Germany and TERMAN in U.S.A. The TERMAN method is presumably the one chosen in most countries.

To begin with, the number of years which the examined person was considered to fall behind normal children was used as a gauge of backwardness. A comparison was accordingly made between intelligence age and actual age, and the same degree of backwardness was differently estimated according to the actual age of the person concerned. We now have got rid of this inconvenience by the general adoption of the term »intelligence quotient», reached by dividing 'intelligence age' (I.A.) with actual age (A.A.).

Although the test should be looked upon as implying definite progress by providing an objective measurement, the weaknesses inherent in the method should be kept in mind. The most obvious of these shortcomings is the failure of the tests to chart factors such as those dealing with energy, industry and temperament, which certainly play an important part in real life. Further discussion of the problems from this aspect would, however, carry us too far.

The results arrived at in a few investigations in this field may be quoted.

HEALY & BRONNER (1926) attach great importance to the general similiarity of their two separate series from Chicago and Boston, because the examinations were carried out by different psychologists and subjective judgments could accordingly be ruled out. In their total material of 4 000 cases over 70 per cent were of normal intelligence, no difference being observed between boys and girls, 10—18 per cent were clearly feeble-minded, and 8—12 per cent mentally subnormal, i.e. intellectually on the border-line between normal and feeble-minded. The numbers of psychoses are different in the two towns, the

figures from Boston and Chicago being respectively 1 and 7 per cent. The difference, however, may be due to random variation. The fact that psychotic patients have been included in this material is, however, remarkable. Diseased persons certainly ought not to appear among a clientele of delinquents and their inclusion can only be ascribed to oversight. 'Psychopathic personalities' were reported only in the series from Boston, the rate being about 3 per cent. This group is small compared with the findings of other workers, a circumstance which the authors attribute to their method of basing the grouping on real examinations instead of, as is usually the case, on the occurrence of repeated misconduct. The authors reach the conclusion that the prevailing belief that the majority of the delinquents consist of feeble-minded or insane has not been substantiated, although the investigation has shown a much greater proportion of mentally abnormal among this clientele than is found among the general population.

GLUECK & GLUECK (1930) have taken great pains in examining the mental habitus of their clients as thoroughly as possible, employing the Binet-Simon intelligence test series. The results obtained were subsequently compared with the school marks. It was found then that only 33 per cent (154 out of 466) were intellectually normal, whereas the dull, border-line and feeble-minded cases numbered 67 per cent (312 out of 466). As psychopathic subjects were considered 17.7 per cent (68 out of 384) while 69 per cent (265 out of 384) were not known to be mentally afflicted. In addition, cases of epilepsy, alcoholic delirium and so on were registered. Roughly, the same results were obtained in a later investigation (1934), when the authors report less than half of the entire number as intellectually normal, i.e. 41.6 per cent (407 out of 979). Although the delinquents admittedly spring from socially and economically lower classes, the authors do not consider this circumstance to account for the palpable difference between their intelligence level and that displayed by the controls made up of 3 500 elementary school children, among whom 79 per cent were normally gifted. Psychiatric examination failed to reveal psychic peculiarities in 443 out of 1 000 cases. Mental disease (psychosis) was found

Table 10. Intelligence level in delinquents according to different authors.

Author	Year	Country	Total number of cases	Intelligence						Psychopathia	
				Normal		Dullness		Debile			
				No.	%	No.	%	No.	%	No.	%
Boys											
1. Lund	1918	Sweden	743	497	66.9	183	24.6	63	8.5	109	14.7
2. »	1918	»	175	116	66.3	31	17.7	28	16.0	38	21.7
3. Nordal ...	1935	»	110	61	55.5	36	32.7	13	11.8		
4. Fritzell ...	1935	»	822	551	67.0	180	21.9	91	11.1		
5. Jonsson ...	1944	»	3352		61.6		6.6		13.5		
6. Arctander	1936	Norway	345	139	40.3	98 [1]	28.4	32 [2]	9.3	117	33.9
7. »	1936	»	232	71	30.6	36 [1]	15.5	15 [2]	6.5	67	28.8
8. Wimmer ..	1929	Denmark	213	102	47.9	57	26.8			54	25.4
9. Gruhle ...	1912	Germany	105	58	55.2	27	25.7	20	19.0	58 [3]	55.2
10. Burt	1925	England			65.1		26.9 [4]		8.1 [5]		
11. Healy & Bronner	1926	U. S. A. Chicago			77.1		9.0 [6]		10.1 [7]		
12. Healy & Bronner	1926	» Boston I			75.0		10.5 [6]		11.2 [7]		2
13. Healy & Bronner	1926	» » II			71.7		8.9 [6]		15.4 [7]		3
14. Glueck & Glueck	1930	»	466	154	33.0	216 [8]	46.4	96	20.6		
15. Glueck & Glueck	1934	»	979	407	41.6	444 [8]	45.4	128	13.1		
Girls											
1. Lund	1918	Sweden	106	78	73.6	24	22.6	4	3.8	13	12
2. Fritzell ...	1935	»	345	195	56.5	98	28.4	52	15.1		
3. Ahnsjö ...	1941	»	550			247	44.9			257 [9]	46
4. Jonsson ...	1944	»	1061		56.8		8.6		16.2		
5. Arctander	1936	Norway	110	27	24.5	27 [1]	24.5	4 [2]	3.6	32	29
6. »	1936	»	180	38	21.1	44 [1]	24.4	10 [2]	5.6	76	42
7. Wimmer ..	1929	Denmark	59	11	18.6	25	42.4			23	38
8. Burt	1925	England			58.2		29.8 [4]		6.8 [5]		
9. Healy & Bronner	1926	U. S. A. Chicago			70.0		6.4 [6]		18.1 [7]		
10. Healy & Bronner	1926	» Boston I			69.9		12.0 [6]		13.5 [7]		3
11. Healy & Bronner	1926	» » II			73.2		5.0 [6]		18.0 [7]		2

[1] Below normal (»undermålere»).
[2] Mental class children (særskoleelever).
[3] Psychopath (»psychische auffällig»).
[4] I.Q. = 70—85.
[5] I.Q. = 50—70.
[6] Subnormal intelligence.
[7] Feeble-minded.
[8] Dull + borderline cases.
[9] Including cases with defective intelligen

only in 3 instances. Accordingly three fifths of the examined boys showed marked mental disturbances of various types.

BURT (1925), testing his material according to Binet-Simon, discovers 64 per cent of normally endowed children, with an intelligence quotient of 85>100 and in addition, 28 per cent of natural dullness and 8 per cent mental deficiency. The author points out that his figures do not compare favourably with those quoted by American workers — in particular as regards the number of mental defects — and submits the explanation that he himself has defined this conception in a narrower sense when employing the line of demarcation generally adopted in England in certifying children for the special school. In any case, mental deficiency was found to be five times as frequent among delinquent children as among the school population as a whole. In the authors' opinion mental deficiency is undoubtedly a notable factor in the production of crimes.

According to the author, a certain relationship can be discerned between the degree of intelligence reduction and the type of delinquency. Cruelty, vandalism and vagrancy are thus characteristics of the very dull-witted, who also particularly often yield to the urge to start fires. The not very gifted are usually temperamentally passive, and become willing tools in the hands of their more enterprising comrades. Active participation only takes place at a more advanced age. Girls reverting to sexual misdemeanours belong to the most advanced of the feeble-minded. In addition, the deficiency of intelligence met with in dull-witted girls is frequently found to be combined with such temperamental peculiarities as make it difficult for them to stand the kind of work suited to their intellectual faculties and paid in accordance with them.

CRÉMIEUX, SCHACHTEL & COTTE (1945), who report on the latest experiences in this field, have examined 2 524 boys and 440 girls, all of whom had been in contact with the legal authorities in Marseilles. In registering the results of their intelligence examinations the headings 'normal', 'sufficient' and 'debility' of different degrees are used. When adding the corresponding figures, 49.2 per cent of the boys and 43 per cent of the girls were found to be of normal intelligence. Strikingly

low intelligence was met with in 8.2 per cent of the boys and 9 per cent of the girls. Between these extremes came slighter forms of debility. The authors do not use intelligence quotients, but only the subjective terms referred to above. For this reason it is hard to form an idea of the real situation. When dealing with other mental aspects comprehended under the heading 'troubles caractériels' the authors are guilty of the same vagueness. Out of the original 3 000 cases only 288, of whom 191 are boys, were left for this investigation, because a considerable number, whose minor peculiarities of character were considered to be due to the incompetence of the parents as mentors, were deliberately left out. Of these 228 cases (46.4 per cent) were normally or sufficiently gifted, whereas the rest exhibited departures from the normal to different degrees. As such were counted various traits of character, for instance, a lying disposition, vanity, impetuosity and so on. It is impossible not to be very sceptical of the results of investigations in which subjective evaluation and haphazard selection have been allowed so much scope.

German authors have devoted considerable efforts to the mental examination of the children taken into custody, and are inclined to look upon abnormal psychic variations as the fundamental causes of social misadaptation.

The older German literature on the subject of delinquents reveals excessive variations in the figures for 'mental inferiority' i.e. between 9.1 and 80 per cent (quoted after Gruhle).

GRUHLE (1912), commenting on this, points out that the lowest figures which have been extracted from official statistics are presumably based on insufficient data, while the extremely high figures are due to the circumstance that most of the cases concerned were examined by psychiatric specialists too liberal in deciding on a diagnosis. In Gruhle's opinion, however, at least 50 per cent of the delinquents are mentally abnormal. In his personal investigation 55 per cent were found to show some kind of mental anomaly. In addition to the conditions difficult to diagnose, by the author designated as psychically peculiar, (»psychisch auffällig»), various complaints are included, for instance periodical headaches, attacks of dizziness, disturbance

of sleep and even 3 cases of epilepsy. Plain mental deficiency, or imbecility in the nomenclature employed was found only in 20 cases, or 19 per cent. The author who did not employ any intelligence test method forms his opinion on available information as well as on the personal impressions conveyed by intimate talks with each pupil.

GREGOR (1918), examining 100 boys, found very few who could be considered as mentally sound. Thirteen, however, were placed under this heading, with the reservation that they were hard to distinguish from feeble-minded cases and displayed many traits of an abnormal character, such as indolence and lack of moral balance. The author's general impression of his cases is the peculiar adjustment of the children's willpower and emotional life which constitutes a trait common to them all. Imbecile and feeble-minded made up 17 per cent.

GREGOR & VOIGTLÄNDER (1918), examining girls, obtained slightly better results or 20 per cent mentally sound, 46 per cent psychopathics, and 32 per cent imbeciles and feeble-minded.

REHM (1926) is still more positive when declaring it hardly possible to distinguish between psychopathic and non-psychopathic cases. In many individuals psychopathic traits are found combined with more or less pronounced feeblemindedness. If the examiner has been courageous enough to prononunce a child to be sound, its subsequent development when in custody is sure to reveal surprising signs of a psychopathic personality.

WIMMER (1929), inspecting detention homes in Denmark, found 111 out of 213 boys (52 per cent) to be mentally abnormal; of these an equal number were feeble-minded and psychopaths. Among 59 girls 48 were mentally abnormal (81 per cent), likewise equally divided between feeble-minded and psychopaths.

ARCTANDER (1936), as mentioned before, divided her material into no less than 7 groups, 4 of which were made up of 2 senior and 2 junior groups of boys and girls respectively. For the purpose of classifying the mental equipment of the children a very elaborate grading was used. However, on joining together the categories designated as of normal and good intelligence

respectively on the one hand and, on the other, the subnormally endowed and the pupils from special classes, it will be seen that in the two groups of boys 52 per cent (139 out of 269) and 58 per cent (71 out of 122) respectively were found to be of average intelligence. The corresponding figures from the two series of girls were 46.6 per cent (27 out of 58) and 41 per cent (38 out of 92) respectively. Thus, 50 per cent — boys a little more and girls a little less — were intellectually normally equipped, whereas the rest displayed more or less submental endowment.

In classifying the children's emotional constitution, the author employs, in addition to the designation »psychopathic», certain peculiarities of character as »queer», »eccentric» or »troublesome» and so on. Lumping together these various designations 86.7 per cent (117 out of 135 cases with information) and 85.9 per cent (67 out of 78) in resp. groups of boys were to be labelled as »psychopatic personalities»: In the two groups of girls the same figures are 91.4 per cent (32 out of 35) and 93.5 per cent (86 out of 92) respectively. The number of children exhibiting traits of abnormal temper appears to be surprisingly high, but the author has received her informations from the personnel of the detention homes and not from physicians.

LUND (1918) first tested his material by the Ziehen method, and afterwards checked up by comparing the results with the general impression of each pupil at daily observations over a long period. The author uses the expression »imbecility» for all intellectual deficiencies. Among the worst cases 59 out of 175, or 33.7 per cent, were defective. Boys and girls from ordinary detention homes show slightly better results or respectively 33.1 per cent (246 out of 743) and 26.4 per cent (28 out of 106) of lowered intelligence. Making the reservation that psychic alterations other than intelligence deficiencies are hard to prove objectively, the author quotes the following figures: 21.7 per cent (38 out of 175) as appertaining to psychopathy and 'psychisch auffällig'. In addition 14.9 per cent or 26 out of 175 were at the same time psychopathic and intellectually backward. Only 12 persons, or 29.6 per cent, were mentally

normal. Lund, however, points out that many of the delinquents displayed grave psychic disturbances, bordering on mental disease.

FRITZELL (1935) shows some hesitation in presenting the information received regarding intelligence, but considers his figures to agree fairly well with those found by Lund. He distinguishes between 3 different degrees — namely: good, fair and feeble endowment — and arrives at the distribution shown in Table 10.

AHNSJÖ (1941) registered 44.9 per cent (247 cases) of intelligence deficiencies and 46.7 per cent (257 cases) of psychopaths, either isolated or combined with mental deficiency, in a material of 550 girls. His information came from the respective detention homes, however; only exceptionally was it based on medical examination or intelligence tests. When compared with the frequency of children in special classes as worked out by DAHLBERG (1937) i.e. 1.7 per cent for girls, the number of dull children in this as well as in other Swedish investigations appears to be remarkably high. The high figures given by Ahnsjö are probably due to the fact that most of his cases (87.5 per cent) were inmates of detention homes for abnormal children.

The perusal of previous investigations accordingly indicates a relatively high proportion of intelligence deficiencies among the delinquents as well as among the juvenile criminals, and in addition suggests that, in particular, the different disturbances of willpower and emotional life covered by the term psychopathy appear at a high rate. Several authors (GREGOR, GREGOR & VOIGTLÄNDER, LUND, BURT), however, seem inclined to infer a causal connection between these forms of psychic deficiencies and delinquency, a conclusion which in any case cannot be considered as proved. Every delinquent does not of necessity display obvious deficiencies, but, from another point of view, all delinquents might be considered abnormal inasmuch as they do not behave like normal children, a circumstance which has led them to be taken in charge. Everything depends on the way in which psychopathy is defined, and on how the line is drawn between normal and abnormal mentality.

An investigation conducted by the *Federal Children's Bureau* into the destinies of 1 000 children from special classes in a number of large towns, showed only 14 per cent to have been prosecuted and punished. The value of the risk figure, 14 per cent, is difficult to judge, however, without knowing the corresponding figure for the normally endowed population. Nor is any information given regarding sex distribution or the length of time during which the cases have been followed up.

In another investigation into the prognosis carried out by the *Child Guidance Centre,* entitled: »The success and failures of subnormal problem children», a material consisting of delinquents as well as non-delinquent children, all with subnormal intelligence (intelligence quotient=75), 103 of whom were boys and 86 girls, was analyzed. It turned out that slightly more than half the number (53 per cent) were successful, whereas the other half were either 'failures' or 'doubtful'. They were all of them more than 18 years old at the time of the examination, and had been followed up for at least 4 years. The two halves were subsequently subjected to a comparative analysis of different aspects such as the degree of deficiency, race, physical development, home standard and so on. Good supervision and certain valuable individual traits of character were very clearly reflected in a good prognosis. At least the later finding appears to be fairly obvious.

Another investigation from the same Centre referred to first and old offenders respectively. The cases which were divided into two groups, the normally and the subnormally endowed, did not show any difference as regards prospects between the poorly equipped and their more fortunate comrades. The results were almost identical for the normal as well as for the subnormal group, and the ultimate outcome more favourable for first offenders than for those who had relapsed into crime.

In the investigations now reviewed, a number of purely physical conditions such as certain diseases, epilepsy, chorea, etc. were looked into, in addition to the mental alterations. According to Lombroso the criminal represented a definite type of human being whose external appearance testified to his criminal disposition. Where delinquents are concerned,

physical peculiarities or so-called degenerative signs such as prognathous jaw and sloping forehead which would brand the children as delinquent have also been looked for, but it can hardly be maintained that such signs were conspicuous.

GRUHLE (1912) showed 41 per cent of the boys at the Flehinger reformatory to be physically sound.

BURT (1925) reports 70 per cent of physically defectives and points out that a comparison with well-behaved children shows poor physical development to be one and a quarter time as frequent among the delinquents. As said before, however, his controls are not wholly above reproach. Ten per cent of the boys and seven per cent of the girls examined by him were suffering from disease or deformity of a nature to be construed by the author as the chief cause of the criminal act; with the reservation, however, that although a physical handicap is frequently a contributary cause, it is relatively seldom the only one, or even the most important. Furthermore the author considers certain diseases, and in particular epilepsy, encephalitis and chorea, to be frequently combined with criminal tendencies. Twelve per cent of the cases had suffered from chorea. The organic disease known by experience to bring about the most serious psychic disturbances is, however, lethargic encephalitis, but the author only reports on a few cases of this disease in his clientele. According to the author, there are also other morbid conditions capable, directly or indirectly, of bringing about queer conduct by affecting the mood.

GLUECK & GLUECK (1930) were not able to substantiate any pronounced increase in the rate of morbid conditions: only 33 cases out of 468 or 7.1 per cent of whom in addition only half the number were suffering from serious diseases such as tuberculosis, epilepsy or hereditary syphilis.

LUND (1918) was of the opinion that 25.7 per cent of the boys from the severe detention home could be proved to suffer from poor physical health. Viewing critically the symptoms marked against each individual case, however, the impression is conveyed that most of these come under headings such as anaemia and general debility, at that time the hall mark of the proletarian child. The only noteworthy observation refers to

the dominating position of nervous disturbances such as bed-wetting, spasms, nocturnal pavor and stammering, which together account for 18.8 per cent.

There is no really satisfactory investigation in which the findings from a control material treated in the same way are discussed. The state of health has presumably not played a leading part in most cases. To what extent a contributary rôle can be ascribed to morbid conditions is hard to say without closer investigations.

As a *summary* it can be said that our present knowledge concerning the background of the problem of delinquency among children amounts to this: most of the children belong to the lowest social strata. Home conditions in childhood leave much to be desired. A great number of the homes are incomplete owing either to illegitimate birth or to the fact that the home life has been broken up by divorce or by the death or absconding of one of the parents. Children were frequently brought up in more than one home. When both parents were living together the standard of the homes was often found wanting economically or hygienically. The qualities of the parents meant they were poorly equipped for their task of bringing up children. Supervision was often very deficient and the children were left to shift for themselves. These children accordingly have been allowed to grow up deprived of the sense of security which only a good home and regular family life can engender. In addition, they are genetically afflicted, either by intelligence deficiency or psychopathy, which leaves them with a handicap in the struggle for life.

PROGNOSIS.

When it comes to the outlook for the delinquents a certain discrepancy can be noted between the results obtained in different investigations. It may be pointed out that the results arrived at will of course depend on the nature of the original material. Although delinquency is a fairly well-defined concept, its scope may be more or less restricted in various countries

and at different times. If only serious cases are investigated the prognostic outcome is naturally worse than when milder cases are also included. Different results may also be explained by the cases being followed up through a shorter or longer period. Obviously, the number of failures increase, the longer the time of observation.

A third reason why the results vary is that records are more or less detailed and the cases judged differently, even when the estimate is based on objective facts such as committed crimes, because criminal laws and their application differ in the various countries and at different times. Then, too, legal responsibility is reached at different ages, and so on. Where subjective impressions are relied upon in preference to objective facts, the results will be subject to even wider variation.

Obviously at least, the fate of the delinquents cannot be categorically forecast. The prognosis may change from time to time and differ in different countries. The outlook depends on the general social structure as well as on the special measures taken against the delinquents.

HEALY and BRONNER (1926) have been able to follow the outcome of a part of their huge material, viz. 420 and 400 boys from Chicago and Boston resp. The youngsters were followed up for about 10 years and their average age at the conclusion of the investigation was 25 years. The authors use the term »success» for those who have been engaged in no criminality and without known detriment to society. Conversely, »failures» denoted actual criminality, but also those were regarded as failures, who were an actual drag of the community, i.e. vagrants, excessive drinkers, extreme loafers etc. The prognostic results were very different in the two towns, both as regards the number of offences committed and their gravity. Among the Chicago boys the results were designated 'success' in 39 per cent (164 out of 420) and 'failures' in 61 per cent or 256 cases. The corresponding figures from Boston were 79 per cent 'success' (316 out of 400) and 21 per cent 'failures' (84 cases). Among the boys from Chicago 13 had committed acts of violence and manslaughter, of which serious offences none of the Boston boys was guilty. 39 Chicago boys were professional thieves as

Table 11. **Delinquency and prognosis in boys.**

Author and country	Year	Years of observation	Age when discharged, years	Total No. of cases	Cases punished for crimes, %	Prognosis		
						Good %	Doubtful %	Bad %
Sweden								
Fritzell	1935	< 7 1/4	10—21	741 [1]	22.1	—	—	—
»	1935	2 1/4—4 1/4	10—21	183 [1]	18.0	—	—	—
»	1935	4 1/4—7 1/4	10—21	557 [1]	23.5	—	—	—
Norway								
Daae	1908	< 3	—	810 [1]	14.1	—	—	—
»	1908	< 3	—	383 [1]	16.7	—	—	—
»	1908	1/2—2	—	427 [1]	6.6	—	—	—
Nissen	1916	< 3	14—21	285 [1]	19.3	—	—	—
»	1916	< 3	14—21	182 [1]	12.6	—	—	—
»	1916	< 3	14—21	103 [2]	31.1	—	—	—
»	1916	< 3	14—19	20 [3]	10.0	—	—	—
»	1916	< 3	14—19	156 [4]	7.1	—	—	—
Arctander & Dahlström	1932	2 — > 10	15—40 [5]	1800 [6]	32.9	—	—	—
»	1932	< 3	15—40 [5]	1636 [6]	22.4			—
»	1932	< 10	15—40 [5]	981 [6]	39.9			
Arctander	1936	2 — > 20	> 20 [5]	1118 [6]	45.1	53.6 [8]	17.7	28.7
»	1936	2 — > 20	> 20 [5]	232 [7]	38.8	64.4 [8]	15.9	19.5
Denmark								
Skjerbæk	1929	< 5	16—19	113 [1]	—	42.5 [8]	30.1	27.4
»	1929	< 5	14—16	88 [1]	—	53.4 [8]	25.0	21.6
Germany								
Gregor	1918	1—2	[9]	100 [1]	—	47.0	24.0	29.0
»	1932	5—7	19—21	91 [1]	—	65.9	11.0	23.1
Kluge	1925	< 3	postschool age	36 [1]	—	44.4	8.3	47.2
Fucks—Kamf	1929	14	17—19	65 [10]	75.4	24.6	41.5	33.9
Fischer [11]	1929	4 1/2—5 1/2	23—24 [5]	— [1]	—	58.4	13.6	28.0
Späth	1934	0—7	of school age	92 [1]	—	64.9	—	29.9
Lückerath	1934	2 1/2—6 1/2	—	166	—	75.9	—	24.1
U. S. A.								
Healy & Bronner	1926	7—13	25 [5]	420 [12]	—	39.0	—	61.0
Glueck & Glueck	1934	5	15—21 [5]	905 [12]	—	11.8	—	88.2

[1] Detention homes for ordinary cases.
[2] Detention homes for severe cases.
[3] Industrial school (tvangsskole).
[4] Private homes.
[5] Age when examined.
[6] Cases taken care of by the Child-Welfare Board.
[7] Random cases.
[8] Including cases with minor offences.
[9] Half of the number 'schulentlassene', half 'schuljährige'.
[10] Reformatory.
[11] Quoted from Fuchs-Kamf.
[12] Juvenile Court cases.

against one of the Boston boys. Other offences perpetrated by the Chicago boys were also of a most serious nature, as reflected in the considerably more severe punishments meted out to them as compared with the Boston delinquents, who were frequently let off on probation. The authors do not venture an explanation of the differences, but confine themselves to the recording of facts. Presumably it is obvious to all U.S.A. readers that conditions are worse in Chicago than in Boston.

HEALY, BRONNER, BAYLOR and MURPHY (1931) later conducted another study of great interest, although it can hardly be called a prognostic investigation in the proper sense of the term. The object was to find out what had become of children who were placed in private homes instead of being taken care of in institutions. Naturally ordinary foster-children — i.e. abandoned or poor children — were not covered by the investigation, which dealt exclusively with problem children who had displayed tendencies towards delinquency of different kinds and degrees. The authors point out that these cases should in no way be considered as 'easy' ones, who as a rule are placed under supervision in their proper homes, but rather as 'hard cases' who had been indicted before the juvenile courts more than once. The investigation, which did not aim to ascertain the ultimate destinies of the children, was planned to afford an appreciation of the results obtained by 'private education', or in other words, to answer the question whether the educational work had been successful. Conforming to American usage the results were designated 'success' and 'failures'. 'Success', according to the authors, means that the child immediately or gradually ceased to misbehave, or that its unfortunate traits of character or bad habits have improved to such an extent as to make it fit to be a member of a decent family. The children were kept under observation for a number of years, most of them for many years. The average age at the time of the investigation was 12 years. In all 501 children were examined. Among the normally endowed 190 out of 217, or 88 per cent, were registered as successes as against the 14 out of 20 of the subnormally endowed who came under this heading. In contrast hereto only 45 per cent or 46 out of 102 classified as suffering

from 'abnormal mentality' were 'successes'. Several indisputable cases of psychopathy were included in this group. The authors also correlated their results with several other aspects such as illegitimacy, age at the time of intervention, different types of delinquency — simple or complicated — without being able to prove any conspicuous influence on the results by any of them. Comparing boys and girls of normal intelligence no sex difference was reflected in their distribution between 'success' and 'failures'. Only the group 'abnormal mentality' showed a slight difference in favour of the girls. Subsequently 97 of the normally endowed 'successes' were re-examined after having left their temporary homes, when 85 or 88 per cent were still going straight, whereas 12 or 12.4 per cent had relapsed. No mention is made of the length of time they were followed up.

GLUECK and GLUECK (1930) checked up on 510 young men who had been admitted to the Massachusetts Reformatory School from the time of release on probation until 5 years afterwards. The results, generally speaking, were not too encouraging. Out of the 422 young men who could be traced 333 or 78.9 per cent had relapsed into criminality. In 89 cases, corresponding to 21.2 per cent, no criminal act was known to have been committed. As the Massachusetts School corresponds more closely to our juvenile prisons than to our reformatories, however, this investigation may be left out of consideration here, all the more so because the average age on admittance was more than 20 years. (It is true that 306 were less than 20 years of age but the remainder were between 21 and 36 years old.)

A later investigation into the prognosis by the same authors (1934) deals with an entire delinquent clientele — as contrasted to the selected cases from detention homes and reformatories analyzed by most authors. The material was made up of 923 boys, concerning whom reliable information was collected, and who had been sentenced by the juvenile court in Boston during the years of 1917—1922. The average age of the boys when taken charge of was 13.5 years; when followed up 20 years. Only few, 39 out of 923, were less than 14 years old, the majority being between 15 and 21 at the time when the pro-

gnostic investigation was carried out. The observation period was 5 years. When 18 boys, who for various reasons had not had the possibility of committing crimes, had been deducted, 905 remained for prognostication — of these 88.2 per cent had relapsed. The rest had adapted themselves to a normal way of living. No influence on the results by one or the other of the circumstances enumerated above could be demonstrated. Boys who had benefited by sentence on probation showed a slightly better prognosis; presumably, as the authors themselves point out, because of selection from the start, as only the most favourable cases are treated in this way.

A great number of prognosis investigations have been carried out in Germany. Many of them are purely statistical and only aim at finding out to what extent the children who are taken in charge later reverted to criminal habits. As a rule these investigations deal with a large number of cases from 1 000 up towards 6 000, and might be considered as representative inasmuch as they include all the delinquent youngsters who had become known to the authorities, and are not confined to cases admitted to institutional care. The percentage of criminality varies considerably in the different investigations. Among the male delinquents the rates of successes (Erfolg) varied between 40.3 and 71 per cent. The disagreement ought to be due to the varying periods of observation in different investigations.

Further investigations into the prognosis have been made in Germany by private workers (GREGOR, VOIGTLÄNDER, v. GRABE, KLUGE, REHM, FUCHS-KAMPF, WAGNER, LÜCKERATH, SPÄTH, NEUBART). Their material are made up of children from detention homes and reformatories and are thus to be considered as selected to a certain extent. In addition, most of them are rather small, comprising only 100 cases or less, even when consisting of boys. WAGNER, VOIGTLÄNDER, v. GRABE confine themselves to analysing female cases while two of the authors, — v. GRABE and REHM — have studied mentally abnormal cases only.

ADELHEID FUCHS-KAMPF (1929) has made a prognostic study of great interest from many points of view. Her material was made up of 90 boys who were admitted to the Flehinger Re-

formatory in 1910. Leaving out those who had been called up for military service on their release from the institution, and who were subsequently killed in the war and, in addition, a few who were transferred to asylums, the author had 65 cases left for estimation of the prognosis. Her investigation is not confined only to information regarding criminality but deals also with the social behaviour in general and, in particular, with the development of the character. According to the nature of the reports received on criminal acts, the author divides her material into 3 groups. I. *Non-criminal* including 12 manifest cases and 4 border-line cases who immediately after their release had committed some trivial offence, while subsequently behaving well; Group II *slightly criminal,* numbering 27 cases; and Group III, *seriously criminal,* amounting to 22 cases. These three groups are thoroughly analysed from different aspects, with a view to finding traits characteristic of the respective groups as a whole. The members of Group I, the non-criminal, are considered to be normal as regards character as well as intelligence. Delinquency among the lads belonging to this group had first manifested itself at the comparatively late age of 14—15 years. The serious criminals of group III represent the diametrical opposite. All were 'abnormal personalities' who displayed delinquent tendencies early in life, in most cases when 11 years of age. Group II, the slightly criminal, occupied an intermediate position in the respects mentioned. Generally speaking, the results must be considered poor, only 16 cases, or 24.6 per cent, being 'successes'. OHLAND (1937) in her survey of the works of earlier authors, is inclined to explain this by the facts that the boys sent to The Flehinger Institution were always particularly advanced offenders. Although the investigation, as pointed out, is of a certain interest, the presentation of facts lacks perspicuity. Information such as age at the time of release, the observation period and so on can only be extracted by time-wasting perusal of the casuistics which occupy a conspicuous place in the presentation.

GREGOR's (1918) work cannot be considered a prognostic investigation in the proper sense of the term, nor has it been conceived as such. The material is small, only 100 boys, selected

from a detention home in 1915. The age at admittance varied between 8 and 19 years, and the average stay in the home was one year. On release, accordingly, not quite half the number were more than 16 years old, while the other half were several years younger. The general impression is conveyed that the results, 47 per cent good, 24 per cent questionable and 29 per cent bad, refer more to the personal opinion of the author than to objective facts, at least as regards the 'successful' cases, concerning whom no bad news had been forwarded.

In the yearly reports issued from different detention homes in Denmark, accounts have for some time been published of the subsequent development of the pupils who had left. But a comprehensive prognostic survey was deferred until 1929, when SKJERBÆK undertook the task. His investigation refers to boys as well as girls who from April 1st, 1922, to March 31st, 1923, had left different detention homes. The average age at the time of the investigation was 14—20 years, and the observation period was 5 years. The author employs the usual groupings, 'good', 'less good' and 'bad' development. His results are shown in Tables 11 and 12 but appears dubious to a certain expect. To the group 'less good' are referred those who tripped up on account of insufficient ability, moral or energy, and also those on whom light sentences had been passed. The author also mentions that some pupils included in the results had been discharged almost immediately because they had to serve sentences, but it is not clear to which group they were relegated. The material, however, is far too small to permit of conclusions, especially as the author has received all his information, including that about criminality, from the personnel of the detention homes and not from official sources.

DAAE (1908) carried out one of the first prognostic investigations in the Scandinavian countries. He followed up from the point of view of relapse into crime 810 boys who were discharged from detention homes in Norway during the years 1901—1907. His facts were derived from the files of the Prison Board. Percentages of crime were calculated for the whole material as well as for the two groups in which those were registered who were discharged during the periods of 1901

—1904 and 1905—1907 respectively. Of the 383 boys discharged between 1901 and 1904, 64 or 16.7 per cent had been sentenced within 3 years, whereas only 28 or 6.6 per cent of the 427 making up the later group, who had left the homes in 1905—1907, had come in conflict with the law. The observation period in the second instance was rather short, maximum 2 years. The author is a pioneer in quoting the percentage of crime corresponding to each year. Accordingly, 20 out of the 64 failures belonging to the first group or 32.3 per cent had relapsed during the first year, 23 or 32.8 per cent within 3 years. His material must be considered as fairly representative. Presumably, however, this is a matter of severe cases, because only this category was taken charge of at the time. The cases have been followed up only for a very short time, a circumstance liable to detract from the value of the findings — the more so because no mention is made of how the non-registered cases were dealt with. The suspicion arises that particularly recalcitrant cases of the vagrant type escaped investigation. Finally, no information is given about the age of the boys at the time of their discharge and consequently it is not known if they had attained the age of legal responsibility.

Ten years later, DAAE's study was followed by a prognostic investigation by NISSEN (1917) including both boys and girls. In contrast to Daae, who treated his material as a whole, Nissen divided his into age groups, and was in this way able to sort out those who had not attained the age of legal responsibility at the time of being discharged. This is certainly an improvement, but some of the children who are excepted from examination, presumably reached the age of legal responsibility during the 3 years of observation, and ought therefore to have been included in the material studied. The maximum time of observation is 3 years. The figure arrived at by the author (55 punished out of 285 or 19.3 per cent) is slightly higher than that quoted by DAAE (16.7 per cent). The author explains the difference by saying that he included delinquents to whom probation was meted out, which Daae had not done.

ARCTANDER and DAHLSTRÖM (1932), also working in Norway, quote considerably higher figures than those of the two last-

Table 12. **Delinquency and prognosis in girls.**

Author and country	Year	Years of observation	Age when discharged, years	Total No. of cases	Cases punished for crimes, %	Prognosis		
						Good %	Doubtful %	Bad %
Sweden								
Fritzell	1935	2 1/4—7 1/4	10—21	322[1]	7.1	—	—	—
»	1935	2 1/4—4 1/4	10—21	101[1]	4.0	—	—	—
»	1935	4 1/4—7 1/4	10—21	217[1]	8.8	—	—	—
White	1940	2/12—6	14—17[7]	83[2]	2.4	—	—	—
Ahnsjö	1941	1—15	6—18[7]	1531[1]	6.7	—	—	—
»	1941	1—15	15—21[7]	392[3]	13.0	—	—	—
Norway								
Nissen	1916	3	14—19	139[1]	2.9			
»	1916	3	14—19	95[1]	2.1	—	—	—
»	1916	3	14—21	44[4]	4.5	—	—	—
Arctander & Dahlström	1932	2—>10	10—>20	962[5]	4.8	—	—	—
»	1932	<3	10—>20	836[5]	3.0	—	—	—
»	1932	<10	10—>20	436[5]	7.6	—	—	—
Arctander	1936	2—>20	—	110[6]	4.5	78.1	14.3	7.6
»	1936	2—>20	15—>40	180[1]	16.1	67.3	21.4	11.3
Denmark								
Skjerbæk	1929	—	19—20	43[1]	—	64.7	26.5	8.8
»	1929	—	16—17	18[1]	—	57.1	35.7	7.1
Germany								
Voigtländer	1918	1—2	—	97[1]	—	63.9	21.6	14.4
Grabe	1923	8—10	—	59[1]	—	64.0	14.0	22.0
Neubert	1927	12—14	—	76[1]	—	40.8	36.8	22.4
Fischer	1928	4 1/2—5 1/2	23—24[8]	—[1]	—	62.2	10.9	26.9
Wagner	1936	3—12	—	127[1]	—	78.9	—	21.1
U. S. A.								
Healy & Bronner[4]	1926	7—13	—	255[9]	—	54.1	—	45.9[10]

[1] Detention homes.
[2] Primary detention home.
[3] Reformatory.
[4] Detention homes for severe cases.
[5] Cases taken care of by the Child-Welfare Board.
[6] Industrial school (tvangsskole).
[7] Age when taken care of.
[8] Age when examined.
[9] Juvenile Court cases.
[10] Of these 19 % prostitutes.

mentioned authors, or 32.9 per cent, corresponding to 592 punished boys out of 1800. Their investigation is distinguished not only from other Norwegian works but also from a great number of other studies, by the uniform documentation of their material which has been collected directly from the files of the Child Welfare Board. The investigation includes 3 347 children who were taken care of by the Child Welfare Board in Oslo during the period 1900—1928. Of these 1800 boys and 962 girls were left for prognostic studies. The difference between the figures presented by NISSEN and these authors, or 19.3 and 32.9 per cent for boys, amounts to 13.6 ± 2.6 per cent and may presumably be accounted for by the different sources employed by the respective authors. Thus NISSEN derived his information from the criminal register which is narrower in scope than The Police Gazette from which ARCTANDER-DAHLSTRÖM obtained their facts. But the considerable difference can hardly be explained only in this way. Arctander and Dahlström might be correct in attributing the incongruity observed to the fact that their material was made up exclusively of children from Oslo who had always been considered as particularly unmanageable. It is more likely, however, that the disparity between the results is due to differences in the length of observation periods — 3 years in the Nissen investigation as against 10 years in the analysis reported by ARCTANDER-DAHLSTRÖM. Naturally, the number of punished individuals increases in proportion to the time elapsed. When picking out from the ARCTANDER-DAHLSTRÖM material the number of boys who were sentenced within 3 years after their discharge, the rate of relapse is shown to be 22.4 per cent (367 out of 1 636), a figure which appears to be more correct and probable. The difference now amounts to 3.1 ± 2.6 per cent. The chief aim of the investigation, however, was to find out whether the outlook was improved, when a child was taken charge of early. 31 boys out of 252 who had been taken care of before the age of 8 years had been punished, making 12.3 per cent as against 36.2 per cent (561 out of 1 548) among those who were over 8 years at the time of intervention. The difference, 23.9 ± 2.6 per cent, is significant and should be conclusive but for the rather dubious starting-point. The

authors have not made any allowance for the fact that their material also includes cases who had been taken charge because of bad home conditions, and where the children themselves were not delinquent at all. This category of children are frequently taken care of at an earlier age than are the delinquents. Without actually lending their colour to the material, they are so numerous as to have some effect on the prognosis. On this account the prognostic result must be considered questionable.

A few years later ARCTANDER (1936) worked up the same material again. In reply to criticism offered from several parts, the material was divided according to the chief reasons for intervention, employing the terms »bad home conditions», delinquency in the child, and both these conditions combined. Still, after this division, those who were taken in charge when 7 years of age were found to have a better chance. The percentage of sentenced boys in the <7 years group was found to be 12.1 per cent (19 out of 157) as compared to 26.8 per cent (60 out of 224), making a difference of 14.7 ± 3.9 per cent. It is open to question whether the significance of this difference can warrantably be construed into the sense desired by the author, because it is hardly possible to decide if a child of less than seven years is delinquent or not.

A detail worth while investigating as a check would be an examination of siblings who had not been in contact in any way with the Child Welfare Board. On doing so, the percentage of sentenced was shown to be higher among the children examined, than among those who were not taken charge of, or respectively 24.1 (288 out of 1 194) and 19.9 (65 out of 327) per cent. The difference amounts to 4.2 ± 2.5 per cent. The author makes the comment that the result does not indicate any favourable influence by the change of environment, but she rightly adds that the children not taken charge of presumably represented less serious cases. In this analysis, however, the author did not separate boys from girls; nor does she report on the quality of the home environment in which the siblings were left.

To FRITZELL (1935) we owe the only Swedish investigation into the prognosis for boys. His material numbers 1 167 cases,

822 boys and 345 girls, who were discharged from detention homes during the years of 1928—1931. Criminal records were obtained from the official files, later supplemented in regard to vagrancy by perusal of the Police Gazette. In the first place all persons who were admitted to reformatories, asylums or mental hospitals were left out. In principle, no objection can be raised to the elimination of the insane and imbecile. It might perhaps be open to doubt, however, whether all the individuals thus weeded out really always belonged to this type. Anyhow, the removal of those who were sentenced to reformatory is a different matter, because the material then becomes misleading.

In common with most previous authors, FRITZELL has not calculated the percentage of criminality corresponding to each year of observation, using instead observation periods of from under 2 to 7 $^1/_4$ years, a less satisfactory method in view of the great variations. In the total group the percentage of criminality among boys was 22.1 per cent (164 sentenced out of 741). Subsequently the author has divided his material into different groups with 2 $^1/_4$—4 $^1/_4$ and 4 $^1/_4$—7 $^1/_4$ years of observation respectively and worked out the percentage of the punished in each group. A little less than half of the convicted had fallen foul of the law within 2 years, and before 4 years had elapsed most of them had been sentenced, i.e. more than three-fourths. The author, however, has calculated among »the punished» 14 cases who were sentenced for vagrancy and 5 cases for drunkenness; both types of misdemeanour are not considered as criminality in the common sense. Furthermore he has failed to pay sufficient attention to such circumstances as if, or when, the discharged persons reached the age of legal responsibility. A number of children were only 10 years old when discharged. This might be of less importance where the girls are concerned, as there were only 13 between 10 and 15 years of age at the time of discharge, whereas the boys in this age group numbered 48.

Another investigation of great interest in this connection, although not a prognostic study in the accepted sense of the term, is the report on relapse into crime in Sweden submitted by DAHLBERG and LINDBERG (1944). This investigation de-

monstrates the age factor to be of great importance with regard to the risk of relapse, which was shown to be substantially greater among persons who were convicted of crimes when young. This, of course, is in perfect agreement with the fact that the risk of committing a first crime is particularly great in the younger age groups (DAHLBERG 1943). 29 per cent of the individuals between 18—20 years of age who, during 1929 were convicted of first offences and filed in the Penal Register, relapsed within 10 years. The corresponding figure concerning first offenders over 40 years of age was 11 per cent. Among those between 15—20 years who received a first sentence of imprisonment, 41 per cent relapsed within 10 years, whereas the corresponding figure for individuals over 40 years was 13 per cent. An analysis of the material of convicts released in 1939—1941 conveys the same impression. Thus 25 per cent of delinquents belonging to the 16—20 years age group were released from prison the first time, and 52 per cent of these relapsed, while the corresponding figures referring to the age group over 40 years were found to be 3 and 8 per cent respectively. The outlook for young criminals, according to the conclusions to be drawn from the 1929 material too, is particularly gloomy as to repeated relapses. 59 per cent of the persons between 18 and 20 years who committed two separate crimes were found to relapse within 10 years, whereas 27 per cent represents the corresponding figure for individuals over 40 years. Age is also reflected in a corresponding way among those who relapsed after having served three terms of imprisonment. Among such individuals in the age group of 21—25 years 73 per cent relapsed, as against 56 per cent in the age group of 26—40 years. According to another noteworthy observation the risk of relapse is particularly high among persons who had displayed asocial signs at an earlier date, *i.e.*, been sentenced on probation or to reformatory education or forced labour, or warned because of vagrancy. Of those subjected to any of the mentioned corrective measures who served terms of imprisonment during the period of 1939—1941, 26 per cent relapsed within 3 years. The figure referring to prisoners not previously examined was 8 per cent. The corresponding figures relating to ex-convicts who

had served terms of hard labour were 45 and 20 per cent respectively.

Prognostic investigations of female cases are fewer and less extensive than is the case with males. Some authors worked up a fairly comprehensive material (AHNSJÖ, HEALY & BRONNER, FRITZELL, ARCTANDER & DAHLSTRÖM, ARCTANDER) but most of them studied a very limited number, in many instances less than 100 cases (v. GRABE, REHM, VOIGTLÄNDER, KLUGE, SKJERBÆK, WHITE).

Prognostic investigations of girls are far more difficult than is the case with boys. The percentage of criminality in women is relatively low and does not reflect their general attitude towards life in the same way as it does for men. Asocial tendencies in women find other expressions. Sexual laxness, for instance, has nearly always been looked upon as an indication of bad morals and irresponsibility. Accordingly this has been used as a criterion in prognostic analyses of female cases. Most authors, consequently, either confined their analysis exclusively to the occurrence of possible criminality in their material or tried to find out if the girls had reverted to prostitution as well.

The American prognostic investigations are more comprehensive than the European because their authors have taken great pains to collect as much information as possible.

HEALY and BRONNER's (1926) aforementioned investigation also covers a group of girls. When those on whom no information could be gained had been ruled out 255 remained for the prognostic estimation. Of these, 138 or 54 per cent were 'successes' and 117 or 46 per cent 'failures'. The authors, however, have not been content with official information only, but have drawn also on the experiences of the persons entrusted with the supervision of the girls. In this way a number of infringements have been taken into account, which were not brought to the notice of the authorities. Accordingly only 9 out of the 117 failures had been brought up in correctional institutions, and only 19 had been sentenced by the court, although not less than 97 were known to have shown licentiousness, and 22 could be called prostitutes. Such information of a personal nature, however, is liable to be more subjective and less reliable

than that derived from official sources and containing reports on measures decided upon by the authorities. In addition it was shown that a remarkably large number of the girls who had appeared in the juvenile courts for sexual misbehaviour had reformed their ways, the figures being 191 out of 255, or 85 per cent. In the group of 'successes' the corresponding figures were 92 out of 138 or 67 per cent. 111 out of the 117 failures had been guilty of sexual misconduct, and 99 of these had previously appeared in the juvenile courts charged with the same offence. Out of the total of 255 cases 64 had been taken in charge for other reasons, and only 6 of these showed subsequent sexual delinquency. v. GRABE (1914) examined the question of the extent to which a number of girls who were taken charge of in detention homes became prostitutes or turned criminals. He was able to substantiate that 65.3 per cent of the girls had behaved in a way not calling for official commendation during an observation period of 6 years.

GREGOR and VOIGTLÄNDER (1918) concluded their comprehensive monography on the problem of delinquency with a limited prognostic study concerning 97 out of 100 girls who, an account of different types of delinquency, had been brought up in detention homes; post-release records were available. The observation period, however, was rather too short — hardly one year — to permit of any real conclusions with regard to the destiny of the girls. Still the authors consider themselves entitled to report 53.8 per cent of improvements, or 14 cases out of 26 'schulpflichtige' (of school age), and among the 'schulentlassene' (post-school age), better results still, or 67.6 per cent successes (48 out of 71). As the first group of girls had presumably not attained the age of legal responsibility, only the later group can be of real prognostic interest. But the observation period is too short and the material too small to permit of general conclusions.

WAGNER (1936) has followed up 250 girls during an average time of 6 $^1/_2$ years. Of these 33 or 13.2 per cent, had been sentenced for crimes, and a considerable number had become prostitutes. Information on this head was hard to obtain, however, and had been offered only in 129 cases, of whom

30 or 23.3 per cent were considered to have taken up this 'profession'.

SKJERBÆK (1929) has also reported on the destiny of the girls submitted to examination in the survey previously reviewed. His material, however, is very small, including only 59 girls, discharged from different detention homes. Six of them were dead or admitted to various institutions, and 7 had evaded the follow-up. It is rather striking that 30 out of the remaining 48, or 62.5 per cent, were characterized as developing well, whereas only four (8.2 per cent) had gone wrong. *Wimmer,* who analysed the same material from the psychological point of view, is inclined to credit marriage with the good results. Presumably the exact opposite could just as well have been the case i.e. that they got married because of their good behaviour. WIMMER further points out that the development had also been satisfactory in such cases where the girls had been mentally defective. It is probably correct that defects of intelligence and character are less liable to make themselves felt in marriage than when earning one's own living.

All Norwegian authors, with the exception of DAAE, include girls among their cases. NISSEN's (1917) data refer only to criminality. He has two groups of girls, the one comprising 139 girls of between 14—19 years of age at the time when they were discharged from different detention homes in the years of 1908—1911. During an observation period of 3 years, 4 or 2.9 per cent of this group had served sentence. The other group of 62 girls from the big towns, who were placed in private homes, showed a slightly worse prognosis, two or 3.2 per cent being punished within the same period of observation. The figures are close, and the numbers too small to warrant conclusions.

ARCTANDER and DAHLSTRÖM (1932) investigated a far greater material, i.e., 962 girls. Of these, 46, or 4.8 per cent, were punished during the entire length of the observation period which in their investigation was considerable — more than 10 years. The number of girls sentenced within 3 years was 25 out of 836, or 3 per cent, and thus more compatible with the results quoted by the last-mentioned author.

ARCTANDER (1936) supplies more detailed individual information about her cases than do the previous authors. She has two groups, one of 110 girls who had left industrial school (»tvangsskole») another of 180 girls discharged from 'detention homes' (»skolehjem»). The percentage of criminality in the first group was found to be 4.5, or 5 cases. The crimes committed were criminal abortion, vagrancy, receiving of stolen goods, and larceny. In the latter group the results were less satisfactory with 29 sentenced, or 16 per cent. The observation period was the same for both groups: 2—20 years.

In order to obtain a more comprehensive idea of these girls' social conditions than was imparted by the bare figures for criminality, the author has procured information from different quarters. The final results are presented under 9 different headings, but they are not easy of access. In summing up, the author reports a positive development in 82 girls who were brought up in industrial schools (74.5 per cent), whereas 23 or 21 per cent developed in a negative direction. In 5 instances, or 4.5 per cent, the development could not be judged. The results were inferior in the author's second group — girls from detention homes — inasmuch as only 113, or 62.8 per cent, had developed positively, 32 per cent negatively, and the rest were undecided. Most of the girls of the first group of whom information hade been gained (89 in all) were married, the others were employed as servants or shop assistants, and so on. In the second group of girls also, many were married — slightly more than 55 per cent. The destinies of the married were on the whole a little brighter than those of the unmarried, but the difference is slight.

Swedish prognostic investigations of girls have been made by FRITZELL, WHITE and AHNSJÖ.

FRITZELL (1935) followed up 322 girls. Of these 24 or 7.5 per cent had fallen foul of the law. A closer scrutiny of the nature of the crimes of which the girls had been guilty shows that 50 per cent, or 12 out of 24, had been convicted of theft and pilfering, whereas 8 girls had served sentences for vagrancy; the latter — as mentioned before — not criminality in the proper sense. However, thirteen of the girls were less than

15 years and 134 were less than 18 years old and thus, the former had not reached the age of legal responsibility so that neither »warning» nor sentence could be issued for vagrancy for either of these categories.

WHITE's (1940) investigation of girls from Gothenburg is interesting, it is true, in as much as it deals exclusively with a clientele from a big city, but the material is very small. Out of the original, 139 girls only 83 remain, and 10 of these were not considered delinquent but had been taken charge of for other reasons. The age of the girls at the time of their discharge is not given, and in almost half of the cases the observation period is less than one year. Reports on crimes are taken from the files of the Gothenburg police, who presumably only registered cases handled by them. The author's main interest, however, has been to establish the social adaptability of the girls in other respects than criminality.

For this purpose she divided the girls into 3 groups with the conventional headings »good», »less good» and »bad». The results referring to the three groups were 63 per cent (52 cases), 23 per cent (19 cases) and 14 per cent (12 cases) respectively. Considering the short observation period, the result appears to be rather on the optimistic side. In addition, information is missing about 6 cases, who at any rate must be considered doubtful.

AHNSJÖ (1941) has carried out the first prognostic investigation in Sweden of a representative material using a more satisfactory statistical method. Like FRITZELL he relies for his information on the Central Penal Register, supplemented by the notes in the Police Gazette. Making allowance for the girls who had not attained the age of legal responsibility, 1955 remained for the author's prognostic study. Two groups are used, one made up of girls discharged from detention homes, the other of girls from reformatories. The total observation period ranges from 1 to 15 years, but — in contrast to almost all previous authors (ARCTANDER, DAHLSTRÖM, FRITZELL, FUCHS-KAMP), AHNSJÖ does not make computations for the period as a whole. Instead of this each year of observation is treated separately, and the relapses are related to the number of persons who during the greater

part of the year were of age as regards legal responsibility. The risks of judicial reaction are subsequently divided up in separate categories, such as readmissions to institution, conviction of crime and caution or sentence for vagrancy. The study is concerned only with the first crime, the rate of relapses being left uninvestigated. After having appeared once in any of the connections mentioned, the criminal disappears from the material.

An examination of the frequencies of crime shows the risk to be greatest during the 5 first years after the discharge, or 7 per cent. During the following 2 decades the risks fall to 3 and 1.5 per cent respectively. The 3rd year is the most critical for girls discharged from detention homes, with a maximum risk of 2.4 per cent. The risks for girls released from reformatories attain their maximum of 4.7 per cent even during the first year.

The risk of being cautioned for vagrancy during the two first decades is estimated at 3.7 and 3.5 per cent respectively. Later the frequency decreases. Comparing the risk figures referring to girls taken charge of because of sexual misconduct with those for girls found guilty of theft, no difference could be demonstrated. It might be expected that girls taken charge of on account of sexual misbehaviour would when released more often than others try to earn their living as prostitutes and thus be cautioned for vagrancy, but this has apparently not been the case.

AHNSJÖ, looking at the problem from the other side, has planned his investigation to include an answer to the question of how many of the discharged cases had escaped any contact with the law whatsoever. He followed up the girls year by year after their discharge during a 10-year period, and found that 72.2 per cent of those who left the detention homes during the years of 1903—1925 had incurred no measures at all. The result corresponding to the later period of 1926—1937 was not quite as good, showing only 62.5 per cent successes. The figures relating to girls released from reformatories during the two periods were respectively 68.7 and 69.3 per cent.

In Swedish prognostic investigations great attention has been paid to the frequency with which the pupils have been read-

mitted to the detention homes. It is only natural that a proportion of the discharged cases should be returned and such a necessity, of course, might be looked upon as indicating incapacity in the young person to look after himself outside the institution. To use the measure as a real criterion of failure, however, would give to it far too great a significance. We have to remember that the detention homes, especially in the earlier days of their existence, very often had to take back their protegés simply because they had nowhere else to go. Many of them were orphans deprived of protection and care, who looked upon the detention institution as their proper home, to which they returned not only when faced with difficulties in their work but also when visited by adversities through no fault of their own, e.g., illness and unemployment. Occasionally children were taken back because, by accident, they had been placed in unsuitable homes. Formerly the decision about re-entry rested almost entirely with the headmasters, a wider scope being given to subjective points of view, and the rate of readmissions therefore differed for the various homes. Another consideration to be kept in mind, which carries as much weight when deciding on taking back a discharged child as well as assigning detention for the first time, refers to the standard of the child's proper home, whether good or bad. If any prognostic significance whatever is to be attached to the frequencies of readmittance, only such cases ought to be selected as were taken back because of criminal action or asocial behaviour.

In FRITZELL's (1935) investigation the frequency of girls taken back was almost double that of the boys, or 23.4 and 12.7 per cent, respectively. Only 44 per cent of these boys were taken back because of crimes or general bad behaviour, whereas the remainder were readmitted either because of unemployment (16 per cent) or some other, not specified, circumstances. Of the girls 61 per cent went back because of general misconduct, 1 per cent because of criminal action. Applying the reasoning submitted above, the percentages of re-entries become reduced to 5.5 per cent for the boys and 14.5 per cent for the girls. FRITZELL does not give any exact date for re-entries. As a rule they had taken place between 3 months

and one year after release. If we add the figures Fritzell quotes for crime and general misbehaviour respectively as reason for readmission for the girls, the result will be 62 per cent, a figure well in agreement with that found by AHNSJÖ to represent the rate of girls taken back into homes because of offences of their own doing, that is 62.6 per cent. The risk figure referring to readmission during the first year among the former inmates of the detention homes arrived at by AHNSJÖ was 16.9 per cent. During the second year the figure was found to be reduced by $^{2}/_{3}$—$^{3}/_{4}$ of the former. If subjected to the same calculating operations as on FRITZELL's finding, the risk figure during the first year is reduced to 10.5 per cent, or slightly lower than the revised Fritzell figure. The difference might be accounted for by Fritzell's failure to fix the exact length of the observation period. The risk run by the girls released from reformatories of being brought back during the first year was estimated by AHNSJÖ at 9.7 per cent. The author was able to prove that the frequency of readmission was higher during the later than during the earlier period, or respectively 19.3 and 10.7 per cent for the first year under risk. The difference 8.7 ± 2.1 per cent is considered by the author to indicate a more effective supervision, or alternatively a general deterioration of the material, necessitating a closer supervision.

For the purpose of comparison, figures from Norway may be quoted. NISSEN (1917) reported on 7.4 per cent of boys and 2.3 per cent of girls as taken back, mostly because of theft.

Most authors, as mentioned before, have approached the prognostic problem from the point of view of the contact of the individual concerned with the criminal law. Where women are concerned, attention has also been given to the part played by prostitution. In Sweden the law against vagrancy has been applied to prostitutes especially in later years when the special regulations for prostitutes were abolished. Obviously, however, a person need not be a good citizen just because he or she has not been convicted of crime or become a prostitute.

The number of investigations dealing with other aspects of the delinquents' development, such as profession, social position, earning capacity and sobriety, is very limited. Most of the

Swedish and Norwegian investigations of this kind are only concerned with the profession chosen. The information was as a rule supplied by the headmasters, and refers only to the time immediately after discharge, or as long as the supervisors followed up their cases. As a rule no studies were made of official reports, nor have the delinquents been followed up during any length of time. The most searching and complete analysis of these conditions comes from America (U.S.A.).

GLUECK and GLUECK (1930) ascertained the employments found by 399 out of 500 boys at a time when 5 years had elapsed since they left the institution. 134 of them, or 33.6 per cent, were considered to have found good situations, 98 (representing 24.6 per cent) were fairly well employed, whereas 42 per cent (or 167) had to content themselves with pitiful jobs. The epithet »good», employed to distinguish a profession, refers less to its actual quality than to the reports made by employers or relatives concerning thrift and ability to stick to the job displayed by the boys. Among the professions may be mentioned factory hands, painters, engineers, transport workers, office clerks and so on. As »fairly well» employed were labelled those who, although regularly occupied in the same kind of professions as the first group, were less attentive, shirked their duties, periodically indulged in bouts of drinking or were inefficient for other reasons. Accidental unemployment on the other hand was not included under this heading. As persons employed in poor jobs were tabulated those without regular occupation, or those with such casual trades as bootcleaning, peddling, playing the barrel organ, drug running, and so on. The border line between 'fairly' and 'poor' appears, however, to be very hard to define. Occasionally it must be quite a matter of chance if a given case is filed under one or the other of these headings. On the whole it seems safe to sum up the situation by saying that only one third had attained a socially satisfactory way of living.

The testing of the boys' economical status revealed only 10 per cent (43 out of 424) to be 'comfortable', *i.e.*, enjoying settled economic conditions, whereas 51 or 12 per cent were found to be very poor and in need of support. The remaining 330 boys

(78 per cent) were designated as 'marginal', which means that they were just able to make ends meet. These figures, however, are difficult to estimate because the authors have omitted to forward comparative information about the economic state of the general population. Maybe the great majority of the American people are living under similar conditions. No figures are given by the author to illustrate the import of inebriety.

FUCHS-KAMP (1929) has extended her prognostic investigation, reviewed above, to include such items as the professions and ways of living chosen by the discharged persons. It was shown that the individuals belonging to the different prognostic groups employed by the author displayed marked differences of care in their work fairly soon after having left the institution. The majority of group I stuck to their jobs, and earned good reports from their employers. Only a few in this group displayed a certain instability and left one employment after another. Such instability was particularly conspicuous in group III. The persons belonging to it showed tendencies to vagabondage and general vacillation immediately on their release. Group II occupied an intermediate position also in respect of profession and stability.

The first world war, 1914—1918, certainly brought about a revolution in the conditions of life of these persons also. Some time after the end of the war, however, all of the persons in group I had settled down in settled occupations such as tradesmen, artisans, porters or public servants, and carried out their duties satisfactorily. The professions which attracted the persons belonging to group III were on quite a different level. All kinds of causal occupations such as those exercised by peddlers, stevedores, dock labourers and odd job men dominated. The second group still held a middle course, most of its individuals were craftsmen or factory workers and therefore in permanent employment, although in a more modest way than the members of group I. If marriage is to be considered as an indicator of social standing, it was reflected also in this instance. In group I all but one were married, in group II 22 out of 27 and in group III only 9 out of 22 were married.

ARCTANDER (1936), studying her two groups of boys, was able

to show that most of the boys belonging to the category criminal boys (kriminella vergerådsgutter), taken in charge by Child Welfare Board had landed on the lower rungs of the social ladder. More than half their number (194 out of 345) were only casually employed as dock labourers or at odd jobs, or earned their living as mendicants and vagrants. The other half were factory workers, craftsmen, navvies or cardrivers in roughly equal proportions, whereas only few had become office clerks or shop assistants. The author's other group of boys, named representative selection (representativt urval) had shown far better social adaptability. Out of 207, about whom information was obtained, less than one fourth came under the heading 'causal workers and their equals'. Sailors, factory workers and the like were represented by increased numbers, and in particular the group made up by office clerks, foremen and similar occupations could register an increase. Out of 180 girls from detention homes (skolehjemspiker) of whom 154 were followed up, 84 were found to be married, and 44 employed in domestic service. The rest were distributed in various professions as seamstresses, typists, nurses and so on. The married were shown to have developed slightly more satisfactorily than the unmarried. It cannot definitely be said whether the marriages contracted by those girls were more successful than those entered into by non-delinquent girls or if their marriage rate corresponded to that prevailing in the general population, as no comparative figures are available.

Investigations concerning Swedish conditions have been carried out by FRITZELL and AHNSJÖ.

FRITZELL (1935) shows that for the majority of the boys work was found in agriculture or forestry at the time of their discharge. These jobs were later abandoned by about half the number. At the time of examination, however, slightly more than a third remained in this occupation. The next largest group, 17.9 per cent, was made up of factory hands, sailors and the like coming next with 9.2 per cent. Artisans and transport workers followed, with 7.7 and 6.3 per cent respectively. The group of unemployed, loafers and recipients of relief amounted to 7 per cent, being thus appreciably smaller

than was the case in the Norwegian investigation. As to the girls, most of them (60 per cent) were found employed in domestic service after leaving detention homes. At the time of the investigation one quarter remained in service. Slightly more than one third were married (34.2 per cent). The group of loafers, etc. was relatively large (5 per cent) for women.

AHNSJÖ (1941) was also able to show that most of the girls about whom information was available were given domestic work when discharged (50.7 per cent). In addition a number took up farm work (10.9 per cent). At the time of the investigation this distribution had naturally undergone some changes. The group of housewives without outside work was found to have increased year by year. Comparing the marriage rate among these girls with that among the general population, the former were found not to be handicapped in the marriage market, being rather ahead of the latter.

The investigations carried out for prognostic purposes were thus found to have yielded different results. Most authors have dwelt on the aspect of the problem dealt with in the penal code and been able to prove that a comparatively high percentage of the male material had been sentenced. It is to be kept in mind, however, that the material at the disposal of previous authors was made up entirely of children who were taken charge of in detention homes or reformatories. Accordingly it is safe to maintain that only the most serious cases were the objects of analysis. No investigation has been made in Sweden or abroad into the destinies of the mild cases, who as a rule were sent to private homes. Nor has the entire host of children passing through the hands of a Child Welfare Board been made the object of a thorough analysis, *i.e.,* a study not confined only to the delinquent children but also comprising such children as were taken away from their bad homes without being delinquent themselves. The two Norwegian investigations (ARCTANDER & DAHLSTRÖM, ARCTANDER) certainly touch the problem but no distinction was made in the earlier of these studies between the children taken charge of because of delinquency and those who were taken charge of on account of their undesirable home conditions only. In the later and more

searching analysis of the same material ARCTANDER left out this last-mentioned category altogether.

By different legal provisions as well as by the creation of the Child Welfare Boards attempts have been made in Sweden systematically to take care of children who are not properly looked after in their homes, irrespective of whether the neglect is due to parental inability as mentors, tendency to delinquency in the child, or a general decay of the home atmosphere. So it is of the greatest interest to find out the characteristics of a material handled by one of these Boards and to form an idea of the prognosis founded on the results so far achieved. In order to afford a better estimation of these a comparatively broad survey of literature has been given. Naturally a brief review conveys only a hazy idea of the results attained elsewhere. It has been essential to review these in detail — in order to ascertain in what respects they can be compared with Swedish conditions. This kind of investigations obviously ought to be conducted in different countries. It cannot be taken for granted that conclusions arrived at in one country also apply to another. The periods during which the various investigations were carried out must also be considered.

CHAPTER II.

DESCRIPTION OF THE MATERIAL.

The present study is based on material obtained from the protocols of the Malmö Child-Welfare Board for the period 1903—1940 inclusive.

The statutes which during this period have governed the activity of the Child-Welfare Board in matters relating to the present investigation are:

1. Law of 1902 concerning »Education of delinquents and morally neglected children» (in the following designated as the Delinquency Law), which was in force during the period 1903—1925 and which was replaced by the law of June 6th 1924 on the public care of children (the Child-Welfare Law), in force from 1926 inclusive.
2. Law of June 27th 1902 concerning criminal suits relating to minors, in force from 1905 to June 30th 1924 inclusive, and replaced by the law of June 6th 1924, with practically no changes concerning our subject, (in force from July 1st 1924).

The Delinquency Law of 1902 aimed »to provide an appropriate education for children under 15 years whose parents or guardians are so licentious, negligent or incapable that special measures appear to be required to prevent delinquency of the children, or who are so delinquent that the educational means of the home and school are found insufficient for their correction».

According to § 22 in the Child-Welfare Law it is the duty of the Child-Welfare Board to adopt the proper measures concerning:

a) children under 16 years, who in the parental home are maltreated or exposed to serious neglect or other risk to life and health;

b) children of this age who are in danger of becoming delinquent on account of depravity and negligence of the parents or their inability to educate the children;

c) children under 18 years who are found to be so delinquent that special educational measures are required for their correction; and

d) persons between 18 and 21 years who are found to lead a disorderly, idle or immoral life or otherwise show a vicious propensity, requiring special measures on the part of society for their correction.

Point *d* in § 22 of the Child-Welfare Law was added by a law of 1934; it has been in force since January 1st 1935, and its formulation was modified in May 1944.

Where the preventive measures indicated by the Delinquency Law and the Child-Welfare Law prove useless or, if adopted, ineffective, according to § 8 of the Delinquency Law and § 24 of the Child-Welfare Law, it is the duty of the Child's Welfare Board to take care of the child and provide for its appropriate education. For this purpose the Child-Welfare Law introduces the concept »protective education».

According to the law of June 27th 1902, concerning the procedure in criminal suits relating to minors — and the revised law of 1924 on the same subject, the Child-Welfare Board is bound at the request of the state attorney to furnish any information to which it has access about the conditions under which the defendant, under 18 years of age, has lived, and whether he has been known to be delinquent, as well as to mention such circumstances, known to the board, as may be presumed to be of significance in deciding on the most suitable measures for his correction.

In recapitualtion, these laws may be said to provide for intervention in cases where homes fail to take care of the children satisfactorily as well as in cases of delinquent children and, after 1934, juveniles leading a disorderly and licentious life.

As to the character and scope of the present material, the following data may be given here:

The total material comprises 2 346 cases, 1 727 boys and 619 girls.

One part of the material, the *Child-Welfare Clientele* comprising 1 035 boys and 566 girls, a total of 1 601 cases, is made up of children, who in the period of 1903—1940 were removed from their homes by the Malmö Child-Welfare Board and given a protective education according to the Delinquency Law and the Child-Welfare Law. However the material does not include children who have been taken care of exclusively under § 22, clause *a* in the Child-Welfare Law, as these children are not to be looked upon as delinquent or liable to delinquency. Also those children who merely received a warning from the board are omitted from the present investigation, as are children whose parents have received a warning from the board and whose homes have been placed under supervision.

The other part of the material, *the Juvenile Court Clientele*,

comprises 692 young men and 53 girls, a total of 745 cases, made up of offenders sentenced at an age of 15—18 years (after 1934 up to 21 years inclusive), concerning whom the Malmö Child-Welfare Board has given testimony under the laws of 1902 and 1924. The first of these testimonies dates from 1906. Prior to 1926 the Child-Welfare Board investigated all such cases very thoroughly, following an established schedule, with data on the delinquent's home, parents and sibs as well as the defendant himself, his marks at school and his behavior during the school period. The reports of all these investigations are still to be found in the protocols of the board, and they have served as the basis for the studies concerning this group. After 1926, however, such information has been given to the state attorney only in cases where the defendant was already entered in the protocol of the board. Thus, the data available on the juvenile court cases after 1926 are sometimes limited to the information obtainable from the police reports. At the request of the court, however, some of these cases have been submitted to a preliminary investigation by the Protective Bureau of Malmö, and thus supplemented with data from these investigations. In this way the information concerning the domestic conditions of the juvenile court clientele of recent years has been considerably fuller than would otherwise have been possible. It has been of particular value that a copy of the final certificate on leaving school has been available in most of these cases.

It should perhaps be pointed out that the Child-Welfare Clientele after 1926, when the age limit for intervention by the Child-Welfare Board was raised from 15 to 18 years and later, after 1935, to 21 years, includes also several cases of sentenced offenders. For, after a conditional sentence or fine, the young offenders were turned over to the Child-Welfare Board. This did not happen prior to 1926, when the age for intervention by the board ended just when the age for the penal authorities began.

The material has since been divided into four groups.

Group I is made up of children who were taken in charge while they were running the risk of becoming delinquent. Thus

the reason for the intervention by the board has been the home, rather than the children. The children have shown no sign of delinquency but have been taken care of exclusively for preventive purposes.

Group II comprises children who have been taken care of on a so-called mixed indication, *i.e.*, the symptoms of delinquency in the child have been so slight that the delinquency in itself would not have justified the intervention of the board, had not investigation revealed such poor home conditions that these, together with the delinquency of the child gave sufficient reason for protective education.

Group III includes all children, regardless of the character of the home, who have been found to be so delinquent that special educational measures were required for their correction. This group further includes juveniles aged 18—21 years, who were found to lead a disorderly, idle or licentious life, and whose correction required special measures on the part of society (§ 22, *d* in the Child-Welfare Law).

Group IV is made up of the Juvenile Court Clientele. It should be pointed out that cases who were first looked after by the Welfare Board and therefore entered in any of the above-mentioned three groups, and who later, at the age of 15—18 years were sentenced in court, are not included in this group.

Concerning the Child-Welfare Clientele it should be mentioned that in the protocols for 1903—1925 no information is given as to which of the above-mentioned indications was the reason for the intervention by the board. Whenever a child was removed from its home reference was made only to § 8, 1, in the Delinquency Law. So the children from this period have been entered in the various groups by the present author. From 1926 inclusive, however, the protocol gives the particular point in § 22 of the Child-Welfare Law, in reference to which each interference has taken place, and the distribution of the children in the various groups has been carried out accordingly.

In view of the unequal upper age limits for the Child-Welfare Clientele during the period covered by the present investigation, the material has been divided into various periods. The first period comprises the years 1903—1925 and includes children

who were taken care of under the Delinquency Law of 1902, in which the upper age limit was 15 years. The other period covers the years 1926—1940. Properly this ought to be divided into two periods, 1926—1934 and 1935—1940, with 18 years for the upper age limit in the former period and 21 years in the latter. In view of the relatively scanty material, however, the two periods will be treated as one. The juveniles whom this rise in the age limit has added to the material from 1935—1940 amount only to 8 boys and 28 girls, so that a division including an additional period can hardly be looked upon as useful.

The information concerning the present material in general has since been supplemented from accessible sources of information, above all from detention homes and from the reformatories. Children who in 1903—1925 were placed in private homes could be followed from one year to another in the inspection record that was kept most carefully and precisely by the teacher, Mr. Nils Silvén, who at that time was child welfare's inspector. This record contains data on the physical condition of the children as well as their mental and emotional characteristics, their behaviour at home as well as in school, their ability to get along at school and the nature of their school reports. I have also received some valuable contributions to my material by direct application to several of the foster-parents.

The individual cases have since been followed up till January 1st 1944 or, in case of death, to the year of death, or — on departure from this country — up to the year of leaving. Individuals who moved from Malmö to some other place in this country have been followed up at their new residences till January 1st 1944.

In order to check the prognoses, data have been obtained from the Central Penal Register, and these have since been supplemented by going through the vagrancy warnings in the Police Gazette.

Data on occupation, social position and civil status have since been obtained, primarily from the Census Office of Malmö, but also from numerous parochial offices throughout the country.

In addition, information has been obtained from the Social

Registers and Temperance Boards in the towns where the persons in question have resided or, if they lived in a village, from the vicar, district council chairman, head of the Temperance Board or the Poor Relief Board in the respective localities.

In order to obtain data as to offences of drunkenness, I have got information from the Central Register of the Board of Control at Stockholm through the State Institute of Human Genetics.

CHAPTER III.

THE INCIDENCE OF DELINQUENCY.

As mentioned in Chapter II, in the present material the Child-Welfare Clientele consists only of children for whom the board has provided a protective education, whereas children, who have merely been the subjects of mild measures — such as warnings — are not included. Even if they have done delinquent acts these may have been casual disorderly acts of a transitory nature. If, on the other hand a child has a tendency to more serious delinquency, it will nearly always be subject to additional measures. So it seems justified to leave out the category mentioned.

As will be noticed from Table 13 and Fig. 3. the absolute figures are subject to considerable variation. During the years prior to the first World War the figures increase, and in the last years of the war, 1917—1918, they show a great rise, but then they fall again rapidly. After this, the figures keep at a relatively low level in spite of the increase in the population.

But the absolute figures are not so illustrative as the relative figures, which take into account the number of children in the various age-groups. In order to give a more correct idea of the situation, these relative figures are recorded. AHNSJÖ (1941) has given relative figures for the entire country in respect of girls admitted to detention homes. He gives the number of girls admitted in relation to the number of girls born in the various years. This method is not altogether correct, as no allowance is made for the fact that a certain number of children die before they reach the age of delinquency, and therefore the resulting figures will be too low — as AHNSJÖ himself pointed out.

Table 13. **Distribution of the cases according to year of intervention of the Child-Welfare Board (or year of sentence).**

Year	Number of cases of the Child-Welfare Board			Number of sentenced cases			Total
	Boys	Girls	Both sexes	Boys	Girls	Both sexes	
1903—05	39	29	68	—	—	—	68
1906—10	151	78	229	40	4	44	273
1911—15	172	76	248	67	5	72	320
1916—20	231	80	311	161	16	177	488
1921—25	85	54	139	56	5	61	200
1926—30	120	69	189	85	6	91	280
1931—35	88	86	174	95	7	102	276
1936—40	149	94	243	188	10	198	441
1903—25	678	317	995	324	30	354	1349
1926—40	357	249	606	368	23	391	997
1903—40	1035	566	1601	692	53	745	2346

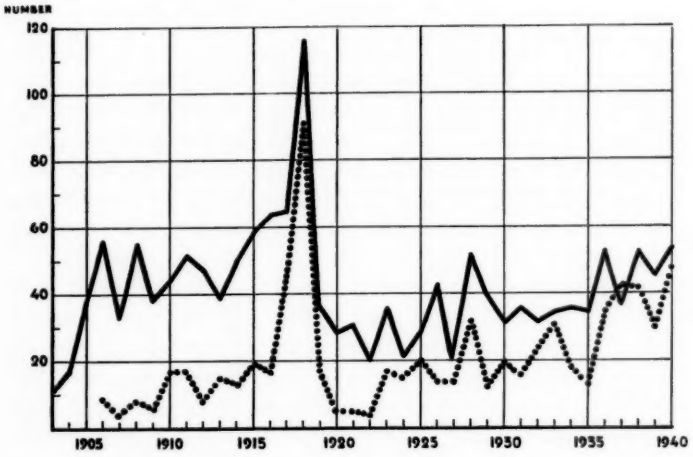


Fig. 3. Number of children taken care of annually by the Child-Welfare Board of Malmö during the period 1903—1940 (groups I, II, and III: whole line) and the corresponding number of cases sentenced in court in Malmö (group IV: dotted line).

Table 14. Number of children taken care of by the Child-Welfare Board and young offenders reported to the Board during the years 1903—40 in percent of the total number of children in the school ages.

Group	Boys			Girls		
	1903—25	1926—40	1903—40	1903—25	1926—40	1903—40
I	0.83	0.54	0.72	0.86	0.53	0.74
II	0.52	0.44	0.49	0.26	0.16	0.22
III	2.0	1.5	1.8	0.45	0.97	0.68
IV	1.7	2.3	2.0	0.14	0.13	0.14

It should be more correct to calculate the incidence of delinquency in the various age-groups and then to calculate the risk of delinquency from the figures thus obtained. For this purpose I have employed the official figures of the number of school children in Malmö at the various ages, correlating thus the number of delinquent children with the total number of children of about the same average age. The outcome of this calculation is given in Table 14.

It is difficult to say anything about the early period. The high figures before and during the first World War might be due to the prevailing social conditions about which it is not now possible to make any definite statement. The inflation during the war and the increased access to ready money undoubtedly played a certain rôle — as did the scarcity of certain goods, particularly in the towns. When the figures for the early period are higher than for the later period this may be due primarily to the great increase during the last years of the war.

The figures for the later period are, I think, of greater interest than those for the earlier period, as they are more typical and are based on a comparatively stable series of figures. Except for Group IV, criminal juveniles, the figures in respect of boys are on an average lower in the later period. It is difficult, however, to decide why Group IV differs from the other groups. As mentioned in Chapter II, this group does not contain all the juveniles indicted in this period. Those already entered in the present Child-Welfare Clientele who later, at the age of 15—18 years committed some criminal offence are not included again

in this group. Furthermore, it cannot with certainty be assumed that all cases of juvenile criminality were referred to the Child-Welfare Board for a statement, nor that this procedure has been practised to the same extent throughout these years. Of course, the rise in these figures may signify an increase in the juvenile criminality in eo ipso, but it may also be due to a change in administrative practice. Without any special investigation it is difficult to say anything definite about these figures, which are recorded merely for the sake of completeness.

From the figures for the later period, however, it is evident that the frequency of *boys* taken charge of because of bad homes has been at least 1 %. In Group I, comprising children who were taken care of only because the home exposed them to a risk of delinquency, the incidence amounts to 0.54 %. In Group II, minor delinquency and bad homes, the frequency amounts to 0.44 %. In other words, these two groups make a total of 0.98 %. These figures are minimum figures for boys taken charge of because their homes have not been satisfactory, as it may be assumed that not all boys coming from such homes are found and taken care of by the Child-Welfare Board.

The next question is how high the frequency of delinquent children may be taken to be. The clearly delinquent are entered in Group III, in which the boys amount to 1.5 % of the boys of corresponding age. If under this heading we want to reckon also the relatively slightly delinquent children, Group II has to be included too, making nearly 2 %.

Employing the same calculation, it will be noticed that in the later period 0.7 % of the *girls* were taken care of chiefly on account of poor home conditions (Groups I and II). In comparison with the corresponding figures for the boys, this frequency is rather lower, because there are relatively fewer girls in Group II, which comprises those children taken care of on mixed indication. Thus the lower ratio for the girls indicates that the boys are more often taken charge of for delinquency than are the girls. This fact becomes even more conspicuous when we consider Group III, which comprises markedly delinquent children. Here the frequency among the girls is about 1 %. Reckoning also the mild cases in Group II,

the total figure increases to 1.1 %, showing considerably lower figures for the girls than for the boys.

It seems safe to say, then, that the incidence of delinquency among girls in Malmö is lower than among boys. This, indeed, is quite in keeping with the fact that criminality is lower among women than among men. (DAHLBERG 1943.) On the other hand, there is a considerable shift in the proportion of the two sexes: instead of one woman per 10 men, we here have one girl per 2 boys.

CHAPTER IV.

AGE WHEN TAKEN CARE OF.

The upper age limit laid down by the Child-Welfare Law for intervention by the Child-Welfare Board naturally partly determines the age distribution of the children and young people taken in charge by the Board. Before 1925 the legal upper age limit was 15 years both for delinquent children and for those taken charge of because of bad homes. After 1925, this limit is 16 years for the latter category, while for the delinquents it was raised first to 18 years and, after 1934, to the 21st birthday. The Law fixes no lower limit either for children in danger of becoming delinquents, or for those who are already so.

Before going into the age distribution, a brief survey will be given of the manner in which the Board was notified of the cases. These problems are admittedly of no major interest; nevertheless the age distribution we get depends to a certain extent on how the cases were brought to the attention of the Board.

The Board is notified from two main sources: public authorities or private persons. Needless to say, the children in the different groups may be reported from different quarters according to the direct call for intervention.

In theory, prophylactic considerations demand the child be taken from a bad home as young as possible, preferably immediately after birth. For this to be feasible, however, systematic supervision of all homes is needed, and this is impossible. Such supervision can only be said to be extended to a certain degree to illegitimate children, following the statute as to welfare officers introduced in 1918. We might therefore expect that after this year, at all events, illegitimate children from bad

homes would be reported at an early age. And this is shown to be so in a distribution of children according to birth: the illegitimate belonging to group I have on an average been reported 1 year earlier than the legitimate. No such difference can be established for children from the other groups. This, however, applies to the entire period from 1903 to 1940.

Table 15. **Percentage distribution of the cases with regard to reporting authorities or persons.**

Reported by	Group					
	I		II		III	
	1903–25	1926–40	1903–25	1926–40	1903–25	1926–40
			Boys			
Police	10.2	26.1	18.1	39.4	55.7	50.3
School	11.3	2.9	58.1	21.3	28.3	13.7
Parents or relations	8.5	7.2	9.5	23.0	9.1	20.7
Child-Welfare Board	26.0	39.1	1.9	13.1	3.3	14.1
Neighbours	25.4	17.4	4.8	—	0.5	0.4
Poor Relief Board	15.8	2.9	5.7	1.6	2.8	—
Temperance Board	—	2.9	—	1.6	—	—
Employer	—	—	—	—	—	0.4
Hospital, doktor	1.7	—	1.9	—	—	0.4
No information	1.1	1.5	—	—	0.3	—
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	177	69	105	61	396	227
			Girls			
Police	6.7	27.3	1.9	17.4	25.3	23.8
School	18.0	3.0	50.1	17.4	47.2	10.6
Parents or relations	4.5	3.0	3.8	30.5	16.1	31.2
Child-Welfare Board	19.1	53.0	19.3	21.7	8.0	23.1
Neighbours	30.3	9.2	15.3	8.7	—	5.0
Poor Relief Board	19.1	1.5	7.7	4.3	1.1	1.3
Temperance Board	—	1.5	—	—	—	—
Employer	—	—	—	—	2.3	3.1
Hospital, doctor	1.1	—	—	—	—	1.3
No information	1.2	1.5	1.9	—	—	0.6
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	178	66	52	23	87	160

As regards the other children in group I, the neighbours are the most likely persons to spread information of the bad home conditions and to report the children to the Board. And as Table 15 shows, during the first period this happened in at least $^1/_4$ of the cases among the boys, and nearly $^1/_3$ among the girls. Nevertheless, it goes without saying that not even the neighbours always learn about the children's dilemma as long as they are infants, but have to wait until they are large enough to run about outdoors and come into contact with persons outside the family. Of course, some of the children in group I are reported and taken in charge as a result of some misdemeanour on the part of one of the older siblings, whereby the board got into the track of the home. It has not been possible to find out to what extent this has been the case, since the entries in the records are incomplete on this head.

It is also likely that some of the reports coming from the authorities actually originated from neighbours who, for natural reasons, did not want the information held against them. It is therefore difficult to distinguish between reports from authorities and private persons. The difference is more formal than real. However, as appears from the table, the parents themselves have sometimes reported their children on the grounds of 'risk of delinquency'. Three fathers have notified the Board of children in their charge, because the mother — who had left home taking the children with her — was living a loose life. In 5 cases, on the other hand, the mother lodged a complaint on account of her husband's rowdy and drunken way of life. Children from this group have also been reported by their schools. Most of these, therefore, are of school age, but some not of this age have also been included secondarily. When the police have reported this kind of child, it was usually because the parents have been proved unsuitable for other reasons, such as fighting, immoral habits of the mother, or criminal conduct of either. As can be seen from the table, then, most of the notifications come from public authorities; in the case of group I primarily from the Board itself, its members or officers; it is, of course, impossible to say how far neighbours and those in the vicinity are the actual 'informers'.

Table 16. **Distribution of the cases according to age when taken care of or when sentenced.**

Age when taken care of or sentenced	Boys				Girls			
	Group							
	I	II	III	IV	I	II	III	IV
				1903—1925				
1— 2	6	—	—	—	4	—	—	—
2— 4	22	—	—	—	12	—	—	—
4— 6	30	1	—	—	31	1	—	—
6— 8	36	8	7	—	28	4	—	—
8—10	32	30	47	—	32	9	7	—
10—12	28	30	107	—	31	14	22	—
12—14	20	24	148	—	29	17	29	—
14—16	3	12	87	117	11	7	29	10
16—18	—	—	—	199	—	—	—	18
18—20	—	—	—	8	—	—	—	2
Total	177	105	396	324	178	52	87	30
				1926—1940				
0— 2	11	—	—	—	11	—	—	—
2— 4	13	—	—	—	8	—	—	—
4— 6	8	—	—	—	11	—	—	—
6— 8	5	5	1	—	12	2	—	—
8—10	5	13	10	—	9	1	1	—
10—12	11	18	31	—	5	5	9	—
12—14	10	18	57	—	9	8	11	—
14—16	6	6	82	80	1	7	41	6
16—18	—	1	38	231	—	—	68	12
18—20	—	—	7	52	—	—	24	5
20—22	—	—	1	5	—	—	6	—
Total	69	61	227	368	66	23	160	23

As Table 16 shows, the children in group I (children taken in charge solely on account of bad homes) were in most cases taken in hand relatively late. During the first period no children were taken in charge before the age of 1 year, because it was thought that such young children would not respond to education and could consequently not become 'morally neglected' — to use the words of the legal term giving the Board the right to take charge of such children. This stipulation is altered in the later Child-Welfare Act, as transpires from the fact that

5 children were taken in hand before the age of 1 year in the later period. During this period, however, a lowered frequency is to be noticed, which certainly is due to a change in the Board's procedure; a number of these children thereby coming under the heading 'social care', and consequently falling out of this material. The figures from the period 1903—1925 show — as will be seen in Figs. 4 and 5 — that the number rises to a maximum at an age of about 7 years, afterwards falling slowly. The later period does not include so many cases, which renders the distribution more uneven.

The mean age of the children in Group I at the time of intervention during the first period is rather over 7 $^{1}/_{2}$ years for boys, and 1 year older for girls. The later period shows throughout a lower age for both sexes, particularly pronounced in the case

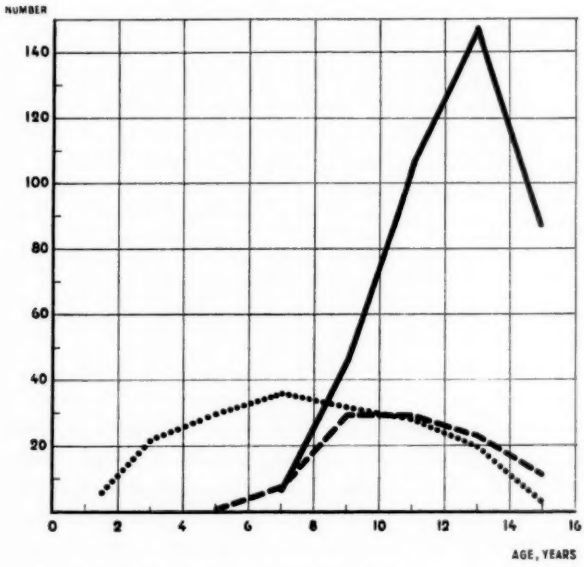


Fig. 4. Age distribution of the boys taken care of by the Child-Welfare Board in Malmö during the period 1903—1925 in group I (dotted line), group II (broken line) and group III (whole line).

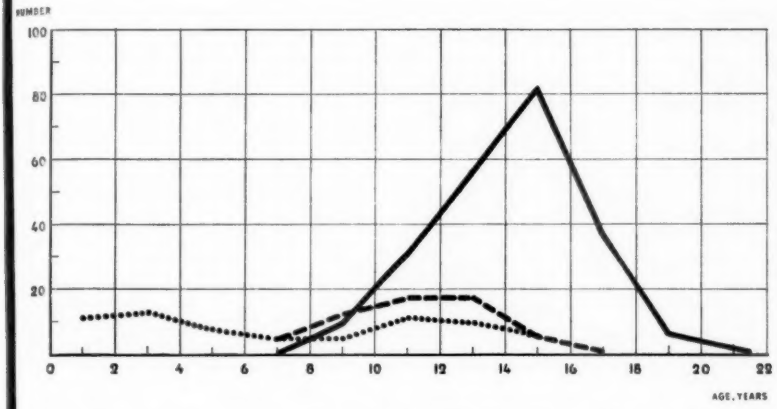


Fig. 5. Age distribution of the boys taken care of the by Child-Welfare Board in Malmö during the period 1926—1940 in group I (dotted line), group II (broken line) and group III (whole line).

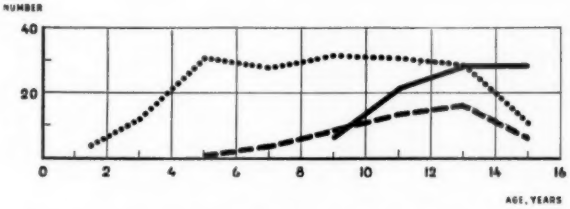


Fig. 6. Age distribution of the girls taken care of by the Child-Welfare Board in Malmö during the period 1903—25 in group I (dotted line), group II (broken line) and group III (whole line).

of the girls, who are now on an average two years younger. This tendency towards earlier intervention is particularly marked when we study the quartile values. During the first period $^1/_4$ of the children were taken in hand before the age of 5—6 years, whereas an equally large number were taken in charge during the later period even before the age of 3 years. In point of fact, most of the children belonging to this category

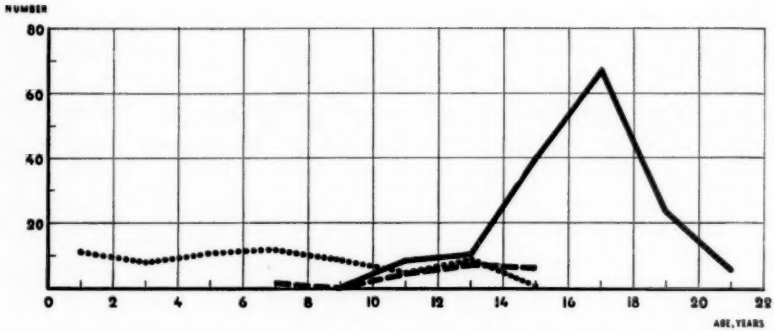


Fig. 7. Age distribution of the girls taken care of by the Child-Welfare Board in Malmö during the period 1926—1940 in group I (dotted line), group II (broken line) and group III (whole line).

should, as already mentioned, be taken in charge young. The fact that they are not is doubtless due to the above-mentioned method of notification, though it is also possible that the home did not deteriorate before the child became older. There are no cases substantiating this, however.

As regards group II, where home conditions also have something to do with the Board's intervention, the lower age limit is to a certain extent set by the age when the subject can embark on misconduct of the kind that can be classified as delinquency — that is to say, about the age of 6 or 7, the attention of the Board being therefore drawn in some way or other to the home in question. The mean for children of this category lies between the ages of 10 and 12 years, and it is quite natural that it is higher than in group I.

As Table 16 shows, maladjustment may prompt intervention from the Board as early as at the ages of 4—6 years. There are only 2 cases of this in the present material, a boy and a girl, both coming under the type 'general delinquency'. As appears from the extract below, the children were taken in charge mainly on account of the bad character of the homes.

$^{17}/_{11}$ Karl, 5 years. Home situated in one of the slum quarters of the town. Father rag-and-bone man, convicted for violence to the police;

drunkard; fined 8 times for inebriation; cautioned by the Temperance Board some years later. Constant squabbling, fights and rows between the parents. 2 children in the home, this boy and one 3 years younger; both neglected and dressed like little ragamuffins. The older was described on intervention by the Board as 'completely delinquent; is utterly wild; spits and snarls; lies and uses bad language'. — Both boys were taken in charge and placed in a good fosterhome in the country; this boy died a few years later of 'unknown cause'; the other developed well and is now living in Malmö as labourer but in poor circumstances.

$^{2}/_{00}$ Petronella B., 5 $^{1}/_{2}$ years. The family moved in from the country some years previously. The father is a labourer; drinks; gives very little money to wife and children. Mother sickly; unable to look after the home. 2 children: Petronella and a 12-year-old sister — bastards of the mother. Both girls taken in charge at the same time, after notification from the older girl's schoolmistress. At the time of intervention Petronella was insolent and agressive, and had begun running about the streets begging. She has been in charge 9 years, is now married and living in the country and as the local vicar says, she is a good housewife giving her children good care.

Passing on to group III (children taken in charge solely for delinquency) we find a shift in the age distribution upwards. When the child's own behaviour has dictated intervention by the community, the frequency rises progressively with age, from 7 years to the upper limit set by the Act for the earlier period. After this time this subject is prosecuted and the rise continues among the Court cases.

However, Table 16 shows that manifestations of delinquency may already be so serious at the ages of 7—8 years that intervention follows for no other reason. It is true that two authors (HEALY, LUND) claim to have had cases of delinquency even several years earlier, but such cases must be extremely rare. There are very few cases of early delinquency in this material: 8 among the boys, and none among the girls.

Of these 8 boys, 5 were guilty of repeated theft, 2 had showed vagrancy tendencies, and 3 'general delinquency'. 4 were illegitimate; 3 of the others were from broken homes; only 1 came from a home where the parents were living together. As regards this latter, $^{13}/_{17}$ *Klas Peter,* 7 years, No. 5 of 6 sibs, nothing is known of the character of the home. The father was a foundry-worker; the family lived in the middle of the town in an ordinary residential quarter. The mother was dubbed unreasonable; took the boy's part. 'If he'd been a rich child it wouldn't have mattered. Such a little boy

doesn't see what's wrong in stealing. The lad was all right till he went to school'. The report was made by the police, who found the boy housed in a staircase, and giving a false name. It was entered on the boy's crime sheet that he had been guilty of various petty thefts, and lived mainly in doorways and staircases. He lied and stole. On one occasion he had lured a smaller boy into a doorway and robbed him of the purse containing money which he held in his hand. The boy who was well equipped intellectually, was now taken in charge and placed in a home in the country, there he remained 7 years. There has since been nothing against him, he is now 34 years old, is married and living in town as moulder.

Not until they reach 8—9 years do the boys show any more extensive delinquent tendencies. The girls reveal such tendencies later, and they do not become very frequent till the ages of 11—12 years.

The later period shows a certain tendency to a shift towards, on an average, higher age groups at the time of intervention by the Board.

Largely speaking, Table 17 shows a shift of the age distribution in the different groups of the material. This is particularly marked for the delinquency clientele proper, where the age difference between the mild cases (group II) and the more qualified ones (group III) is pronounced. During the two periods, the difference between Groups II and III are 1.4 ± 0.23 years and 2.8 ± 0.34 years for the boys, and 1.3 ± 0.37 years and 3.7 ± 0.53 years for the girls, thus being statistically significant for both periods for boys and girls alike. In group III (children taken in charge for more serious delinquency) we find that the mean age at intervention during the first period was 12.3 years for the boys, and 12.8 years for the girls. During the same period, $^1/_4$ were taken in charge even before the age of 11 years, boys and girls alike; during the later period, however, the lower quartile for boys was $12\ ^1/_2$ years and for girls 15 years. During the earlier period the difference in age between the sexes is not so pronounced. The difference between the means is 0.5 ± 0.21 years, while for the later period it is 2.0 ± 0.24 — that is to say, significant for the latter period only. Thus, the mean age when taken in charge was lower during the first period than during the later, for both boys and girls. This is probably due in large measure to the raised upper age limit

Table 17. **Mean age and median age of the cases when taken care of by the Child-Welfare Board or sentenced.**

$M \pm \varepsilon(M)$ = mean ± standard error of the mean.
σ = standard deviation.

Group	Age when taken care of or sentenced					
	No. of cases	$M \pm \varepsilon(M)$	σ	First quartile	Median	Third quartile
Boys 1903—1925						
I	177	7.8 ± 0.26	3.4	5.1	7.7	10.5
II	105	10.9 ± 0.21	2.2	9.2	10.6	12.6
III	396	12.3 ± 0.09	1.9	11.0	12.5	13.8
IV	324	16.4 ± 0.04	0.8	15.7	16.4	17.1
Boys 1926—1940						
I	69	7.4 ± 0.57	4.8	3.0	7.3	11.0
II	61	11.4 ± 0.30	2.3	9.6	11.4	13.1
III	227	14.2 ± 0.16	2.4	12.6	14.4	15.8
IV	368	17.0 ± 0.06	1.1	16.1	16.9	17.7
Girls 1903—1925						
I	178	8.7 ± 0.26	3.5	5.9	8.7	11.8
II	52	11.5 ± 0.32	2.3	9.9	11.3	13.4
III	87	12.8 ± 0.19	1.8	11.5	13.0	14.3
IV	30	16.5 ± 0.16	0.9	15.8	16.5	17.3
Girls 1926—1940						
I	66	6.7 ± 0.50	4.1	3.3	6.7	10.0
II	23	12.5 ± 0.50	2.4	11.3	13.0	14.5
III	160	16.2 ± 0.18	2.2	15.1	16.4	17.6
IV	23	16.9 ± 0.24	1.2	16.0	16.9	18.0

introduced in 1926, which must have affected the girls in particular, since they are often taken in charge for sexual offences.

As regards the criminal juveniles (group IV), finally, all the young people who during the given time have been sentenced are not included in this group — of reason given before. The figures for the age distribution are therefore not so interesting, and are given merely for the sake of completeness.

Summing up, it may be said that when group I ('risk of delinquency') is concerned, the bad character of the home is

as a rule not discovered and proceeded against until school age has been reached. It would naturally be a good thing if it could be discovered earlier, even if it may be that the character of the home may not play so great a part during early childhood as later on. When delinquent children are concerned, on the other hand, the age is higher and rises in frequency until the criminal 'majority' is reached. DAHLBERG's investigation (1943) shows that the risk of being convicted of crime is greatest at the ages of 15—18 years. Probably, the frequency of criminality in a wide sense rises from school age up to puberty. When the subject gets to the age when he can begin working, the frequency falls.

It would naturally be of interest to know whether asocial behaviour began to manifest itself in these children while still quite young. A number of authors have attempted to obtain information as to such manifestations in their clientele (GRUHLE, LUND, GLUECK and GLUECK). Besides the difficulty of reading early manifestations of asocial conduct aright, there are naturally other drawbacks — the question of where the information came from, for one. The statements coming from the children themselves cannot be wholly trustworthy. Children are sometimes apt to be reticent about their earliest exploits, but they may also boast about them. Information from the parents must also be taken with a certain cautiouness. In addition, these data are often vague as regards the establishing of times, and expressions such as 'always', 'since he was quite small' are commonest in the records of the Board. Added to this, misdemeanours at a tender age are, as already mentioned, often regarded with indulgence, and taken to be more mischief and playfulness than outbreaks of delinquent tendencies. Consequently, information of this kind is a very unreliable foundation for an investigation.

All the same the author has attempted to get an idea, when possible based on more objective statements, as to whether there had been any misconduct earlier on. To this end data was collected as to whether the subject had, at an earlier date — i.e. before intervention by the Board — been in contact with the authorities, i.e. the Child-Welfare Board or the police. The

point is that if delinquency of more serious kind had been in question, it should have led to a caution, which is of course entered in the records of the Board or the police annals. Milder manifestations, which could be corrected by the home or the school, have not been looked for, of course. The data could by no means be considered complete, but should be able to give a certain idea.

Children from group I are not included in this investigation, because when taken in charge they showed no delinquency symptoms, measures being taken solely on account of bad home conditions.

After lumping together the data from the different periods, Table 18 shows that half the number of boys in group II (mild cases of delinquency) had previously prompted action from the authorities. Not quite $^1/_3$ were taken charge of within 6 months of the first contact, while nearly $^1/_5$ had shown delinquency even earlier.

Table 18. **Distribution of the cases according to earlier dealings with the Child-Welfare Board or with the police.**

Interval (in years) between first contact and time when taken care of	1903—1940					
	II		III		IV	
	No.	%	No.	%	No.	%
Boys						
0 [1]	84	50.7 ± 3.9	247	39.6 ± 2.0	411	59.5 ± 1.9
< 1/2	51	30.7 ± 3.6	172	27.6 ± 1.8	82	11.8 ± 1.2
1/2—∞	31	18.6 ± 3.0	204	32.8 ± 1.9	199	28.7 ± 1.7
Total	166	100.0	623	100.0	692	100.0
Girls						
0 [1]	51	68.0 ± 5.4	160	64.8 ± 3.0	37	69.8 ± 6.3
< 1/2	16	21.3 ± 4.7	48	19.4 ± 2.5	5	9.4 ± 4.0
1/2—∞	8	10.7 ± 3.6	39	15.8 ± 2.3	11	20.8 ± 5.6
Total	75	100.0	247	100.0	53	100.0

[1] Indicates that the child, before taken care of, has not been in contact with the police or Child-Welfare Board.

Group III (cases of more serious delinquency) shows a still clearer difference for the boys. Nearly $^1/_3$ proved delinquent more than 6 months before being taken in charge.

In group IV (criminal juveniles), finally almost 40 % showed delinquency before prosecution, though most of them (60 %) had not previously been in contact with the authorities, falling foul of society for the first time at the time of the prosecution — this in contrast to the boys from group III, where the converse can be said to obtain, i.e. 60 % with earlier delinquent tendencies and 40 % without.

About half as many girls as boys in group II had shown delinquent tendencies more than 6 months previous to prosecution.

The same state of affairs holds for girls in group III. The percentage of those taken in hand direct is high, as compared with the boys, 65 per cent against 40 per cent for the latter. On the other hand, the percentage showing delinquency more than 6 months preciously is relatively low, compared with the boys, namely 15 against 30 per cent. The differences are statistically significant in both cases.

In group IV, finally, the difference from the boys in the same group is comparatively small both for those girls who had not previously been in contact with the authorities, and for those who had.

Summing up, then, we can say that on the whole the girls in the entire material yield better figures than the boys inasmuch as fewer of them had previously received a caution; this is particularly marked in group III. It may be that the girls are more often amenable to a caution, and therefore require no further measures. It is perhaps more probable that the girls are less liable to commit acts of the kind and degree prompting a caution from the authorities. But it may also be that the Board found the misconduct of the girls of a kind calling for immediate steps. A sense of the risks of sexual offences, which are particularly common among girls, may well have dictated intervention at once. Probably several factors enter the picture.

CHAPTER V.

TYPES OF DELINQUENCY.

It would have been desirable on the basis of personal examination of the individual cases to divide the material according to differences in temperament and according to the psychological motive of the acts. No such investigation has been carried out, however, so the material has had to be divided according to the various manifestations of misdemeanor, asocial or criminal, presented by the individuals, that constituted the direct cause of the intervention by the authorities.

Naturally any attempt to divide material of this kind will to some extent be schematic as well as subjective. Difficulties are encountered in the classification of the delinquent acts of the various individuals, and transitions between the various types are met with continually, as no case is exactly like another.

Still, it would be safe, I think, to claim that as far as delinquency and juvenile criminality are concerned a certain kind of crime and a certain way of action will often be characteristic of juvenile offenders, and that just this specialization — the frequent repetition of the same kind of crime or the same sort of asocial conduct by the same individual — which sometimes may be said to signify a particular tendency in the character of the individual, *e.g.*, vagabondage, makes it reasonable in some degree to speak of different types of delinquency.

Thus the material is first divided into four main groups, crimes against property, vagabondage, sexual misdemeanour, and general delinquency. In addition there are 6 sub-groups, each of which includes two types of the four main groups. Then we have 4 additional sub-groups, in each of which three of the main groups is represented. But the latter have all been lumped

into one group because of the scanty numbers of individuals in the sub-groups. Finally there should be a twelfth group comprising individuals practising all four kinds of delinquency. But no such individuals are found in the present material.

In this grouping only the various kinds of delinquency are taken into consideration, not how often such acts have been recorded in the »black book» of the individual. In this connection it may be mentioned that a single asocial act hardly ever gives rise to intervention by the board. As a rule it requires delinquency of a certain intensity, established by repeated acts, before the Child-Welfare Board intervenes by removing the child from it's home. This applies, of course, only to the children who have been taken care of on account of their delinquency, not to cases where the home conditions have been a contributary or decisive cause of the intervention.

Undoubtedly it would have been of value to get an idea as to the degree of delinquency at the time of intervention by the board. An attempt to obtain this has in fact been made, but failed on account of the vague statements usually available concerning the number of offences committed. In most cases such data are limited to remarks such as »several times», »often», »continually», etc. which do not lend themselves to statistical treatment. Not even with regard to theft has it been possible to obtain exact information as to how often the individual has taken part in such acts. Only when gangs are involved do the data become more definite. But they are relatively few and refer mostly to the crime of theft, while it has not been possible to obtain definite statements about other offences, and hence no tabulation of the material in this respect has been made.

A definition of the various main groups is hardly necessary although their individual scope has to be touched upon. The scope of the term »*crimes against property*» hardly requires any explanation. Qualitatively this heading covers many different sorts of crimes, from petty larceny to more qualified thefts, with or without burglary, as well as forgery, embezzlement and fraud. It is most often a question of petty larceny at home as well as at school, but perhaps even more often in shops or

warehouses where access to the tempting objects is easy. In many cases one might say that the opportunity makes the thief and that lack of control has been a contributory factor leading to the individual's first criminal step. Generally the things that have aroused the desire of the children have been of slight value though such a kind that it was easy not only to take them but also to dispose of them, especially in the first period when less exacting scrap-merchants were willing to receive them. Goods of various kinds stored in the depots of such merchants — *e.g.,* plates of lead, pieces of cables, etc. — were often the goods required. Burglary of attics with thefts of various articles as well as cellar thefts occur relatively often. Stealing of rabbits and pigeons is particularly common. In the later period, »borrowing of bicycles» has become very frequent. Breaking into slot-machines and kiosks selling sweets, which constitute a specially favoured object for gangs, has played a conspicuous rôle in the later period. But, naturally, it is often a question of theft of money, usually relatively small amounts, and not infrequently from the individual's own needy family, though petty thefts of money at places of work also occur. In several cases larger amounts have been stolen, the so-called »errand boy's embezzlement». Embezzlement and fraud occur but relatively seldom, and forgery usually concerns money orders or occasionally cheques. In a sub-group of crimes against property I have reckoned willful damage to property, which has always occurred together with other offences. This damage may be of a relatively mild character as, for instance, to puncture bicycle tyres, and then it should be reckoned rather as mischief; but it may also attain greater proportions — e.g. destruction of growing crops, complete demolition of shop fixtures, or senseless destruction of articles of food in cellars, etc.

Closely related to such wilful damage is arson, which has occurred among the boys in 19 cases, whereas no instance has been recorded among the girls. In no case has this offence been the only reason for intervention by the board, and only in 2 cases did this offence result in a relatively severe damage — as when a boy set fire to a barn »just to see if it would burn» and it burnt to the ground. Another boy caused a big fire while

playing with other boys. In the remaining 17 cases the incendiarism has only been a secondary symptom, though it manifested itself again and again. In this group of boys the youngest was 7 years and the oldest 14 $^1/_2$, while the average age was 11 years. These cases, then, afford no evidence in support of the view that pyromania belongs above all to the age of puberty and is a symptom of psychic lability during these years. They are rather to be taken as a manifestation of an extreme type of lust for destruction as damaging acts of other kind were of frequent occurrence among these boys.

On going through the available information concerning the intellectual level of there 19 boys 8 were found to be of normal intelligence, 2 were backward, and 4 were of poor intelligence. The remaining 5, about whose intelligence no direct remark was recorded, were all in the forms corresponding to their ages so that at any rate they had shown no sign of markedly inferior intelligence. Even though these figures are much too small to allow of any exact estimate, it nevertheless seems evident that these incendiaries are not particularly retarded intellectually. On the other hand, 9 of them presented mental features that undoubtedly were suggestive of psychopathy.

The group of *vagabondage* comprises several categories. It includes cases of truancy in the usual sense of the term, *i.e.*, children who neither by hook nor by crook can be made to participate in school work but prefer to roam about in the streets. Generally the escapades are aimed at districts of the town other than the one in which they are living, and especially the harbour districts, with its various tempting possibilities, has often been a favourite haunt for these loiterers. Those children who from fear or some other motive have not dared to return home but have lived for days and nights in cellars or attics or other stranger places are also placed in this group. A large part of the group is made up of boys who repeatedly ran away from home and stayed away for a few days and nights or wandered about for weeks in the town or in country districts, getting their food by begging, often under some expedient cloak. The group also includes children who have gone on long trips or real tours of larceny that more or less correspond to the

vagabondage of adults. All too often kind-hearted truck drivers have been ready to give these »smart youngsters» a lift when they have run away. Also older boys — »workshy youths» — are reckoned as belonging to this type, which thus has a rather heterogeneous composition. From a social point of view the danger of vagabondage lies in it's great possibilities for other offences such as thieving, begging, vagrancy and liability to demoralisation for the boys, and sexual offences for the girls.

As to *sexual offences,* naturally differences assert themselves between girls and boys in their conduct. The types of delinquency among the girls will be discussed later on. As far as the boys are concerned, the sexual offences dealt with by the board appear to have been of a rather mild character. Especially in earlier days, there seems to have been a tendency to judge sexual offences severely.

The group of *general delinquency* covers all other delinquents. It does not include individuals who have committed any of the abovementioned acts, but merely such children as have shown various kinds of asocial behaviour and various forms of bad conduct and are therefore unmanageable. The most common symptoms are nasty behaviour, impudency, lying and untrustworthiness, contumacy, indolence and negligence, untimely speech, swearing etc. But begging, cruelty to animals and other forms of cruelty are also included under this heading. Obviously, all these symptoms need not necessarily be present simultaneously in a child for him to be labelled a delinquent although a certain intensity of the symptoms present is required. This group also includes acts of violence. There has been no question of more serious kinds of violence, but mostly fights among the boys where various kinds of weapons have been used. Thus, for instance, in a fight one of the boys had used his knife, while another boy had used brass-knuckles. Finally, in this group we also have to include those cases about whom the available information is very meagre.

The various types of delinquency are of course subject to wide variations in frequency. Therefore a survey will first be given of the frequency of the various types of delinquency in

Table 19. **Distribution on different types of delinquency in boys.**[1]

Types of delinquency	1903—1925		1926—1940		1903—1940	
	No.	% of N	No.	% of N	No.	% of N
Group II						
Total number of boys = N	105	—	61	—	166	—
Crimes against property	45	42.9 ± 4.8	43	70.5 ± 5.8	88	53.0 ± 3.9
of these: Malicious damage	3	2.9 ± 1.6	—	—	3	1.8 ± 1.0
Arson	1	1.0 ± 0.9	4	6.6 ± 3.2	5	3.0 ± 1.3
Vagrancy	70	66.7 ± 4.6	30	49.2 ± 6.4	100	60.2 ± 3.8
Sexual offences	3	2.9 ± 1.6	—	—	3	1.8 ± 1.0
General delinquency	33	31.4 ± 4.5	15	24.6 ± 5.5	48	28.9 ± 3.5
of these: Assault (and battery)	—	—	—	—	—	—
Group III						
Total number of boys = N	396	—	227	—	623	—
Crimes against property	331	83.6 ± 1.9	170	74.9 ± 2.9	501	80.4 ± 1.6
of these: Malicious damage	32	8.1 ± 1.4	19	8.4 ± 1.8	51	8.2 ± 1.1
Arson	9	2.3 ± 0.7	5	2.2 ± 1.0	14	2.2 ± 0.6
Vagrancy	204	51.5 ± 2.5	119	52.4 ± 3.3	323	51.8 ± 2.0
Sexual offences	8	2.0 ± 0.7	8	3.5 ± 1.2	16	2.6 ± 0.6
General delinquency	54	13.6 ± 1.7	40	17.6 ± 2.5	94	15.1 ± 1.4
of these: Assault (and battery)	5	1.3 ± 0.6	2	0.9 ± 0.6	7	1.1 ± 0.4

[1] Every case is counted as many times as it belongs to the different types.

which each individual is put down for each of the different types, and thus the same individual may figure several times.

Considering the entire period, 1903—1940, in Group II, cases of slight delinquency, the most common types are crimes against property and vagabondage, with some preponderance of vagabondage. Thus we find that vagabondage occurred in no less than 60 per cent of the *boys* in this group. Property crimes was somewhat less common, being recorded in 53 per cent of the cases, but the difference is not statistically established. As mentioned before, damage to property and arson are included among crimes against property. These two kinds of offence appear to have been fairly unusual, only 2 % of the boys in this group being guilty in this respect. »General delin-

quency» is third among the offences, occurring in 29 per cent of the boys in this group (cf. Table 19).

If we investigate Group III, severe cases of delinquency, for the entire period we find a distinct difference in the frequency of the various types of offence. Here crimes against property are considerably more common than vagabondage, the former being recorded in no less than 80 per cent of the cases, the latter in 52 per cent. In contrast thereto, »general delinquency» is recorded in only 15 per cent of the cases. Wilful damage occurs in 8 per cent of the cases in this group, and thus it is more frequent here than in Group II. Violence as a contributary cause of intervention by the board was not observed in Group II, while in Group III it occurred in 7 cases or in 1 per cent. Arson occurred in about 2 per cent, i.e. equally often among the slight and severe cases of delinquency.

The difference between Group II and Group III in the ratio of the various offences may reasonably be explained as due to the circumstance that, for a child to be taken care of on account of his own achievments (Group III) these must have been considerably graver than those of children in Group II, where the home conditions constitute a contributary reason why the boys are taken care of. Besides it is to be kept in mind that the children in Group II are younger and, consequently, have not been able to attain as much as the somewhat older boys in Group III. This, I think, will also explain why »general delinquency» occurs more often among the relatively slight cases than among the more severe.

Here it may be mentioned that on comparison of the two different periods, 1903—1925 and 1926—1940, with regard to the frequency of the types of delinquency, we find an obvious difference only within Group II. Here vagabondage is more common in the first period, while crimes against property appear more frequently in the later period. Very likely, in the first period a greater importance was attached to vagabondage — i.e., in these cases merely simple truancy — than in the later period, and this is the reason why vagabondage occupies a predominant position in the first period. It is conceivable, however, that the frequency of petty larceny has increased and

Table 20. **Distribution of delinquency in boys on different combinations of types.**

Types of delinquency	1903—1940			
	II		III	
	No.	%	No.	%
1. Crimes against property	36	21.8 ± 3.2	234	37.5 ± 1.9
2. Vagrancy	42	25.3 ± 3.7	63	10.1 ± 1.2
3. Sexual offences	1	0.6 ± 0.6	8	1.3 ± 0.5
4. General delinquency	16	9.6 ± 2.3	26	4.2 ± 0.8
5. Crimes against property + vagrancy	37	22.3 ± 3.2	217	34.8 ± 1.9
6. Crimes against property + sex. off.	1	0.6 ± 0.6	3	0.5 ± 0.3
7. Crimes against property + gen. del.	12	7.2 ± 2.0	28	4.5 ± 0.8
8. Vagrancy + sexual offences	1	0.6 ± 0.6	2	0.3 ± 0.2
9. Vagrancy + general delinquency	18	10.8 ± 2.4	23	3.7 ± 0.8
10. Sexual offences + general delinq.	—	—	1	0.2 ± 0.2
11. At least 3 of 1—4 in conjunction	2	1.2 ± 0.8	18	2.9 ± 0.7
Total	166	100.0	623	100.0

that of vagabondage decreased. For, considering the number of individuals taken care of per year, vagabondage is found to have decreased markedly, while crimes against property have increased (cf. Table 20). As to the other types of delinquency within Group II as well as Group III the distribution is about the same in both periods, on which account it is found convenient here to consider the cases from both periods together.

It may now be of interest to see how many boys commit each type of delinquency. In Table 20 the individuals are recorded while in Table 19 the offences were the statistical units. From Table 20 it will be noticed how many boys commit only one type of delinquency and how many boys commit more than one type. In this connection it should first be pointed out that the type »general delinquency» is rather dubious from this point of view. If a child has been guilty of a real offence it may be that it was not considered worth while to put down such common things as would entitle him to be included under the type of general delinquency. So it may be that the combination

of the other types with general delinquency is not recorded as often as it should be.

If a child is highly delinquent we might expect it to belong to various types. If it is only slightly delinquent it should have qualified in only one way. Therefore, we ought to find more single types in Group II (slight delinquency) than in Group III (marked delinquency); and this is in fact the case. The single types amount to 57 per cent in Group II, 53 per cent in Group III. But this difference is smaller than would be expected. If we leave general delinquency out of consideration, however, and reckon the cases in which it was recorded as single types, the respective figures are 75.3 and 61,5 per cent. So now the difference is greater.

Otherwise Table 20 hardly tells us anything of interest. That property crime and vagabondage are combined relatively often — i.e. in one-fourth of the cases in Group II and one-third of the cases in Group III — is only natural, as both offences are common in this material.

Sexual offences are less common among boys, and only 19 out of 789 boys were found guilty of such offences, 3 of them among the cases of slight delinquency, 16 among the more severe. In view of their scarcity it may be appropriate here briefly to give an account of these cases.

Concerning the 3 boys in Group II, the information given in the records is very scanty. About 2 of them, respectively 8 and 14 years old, it only says that they were guilty of »immoral conduct towards small girls». Concerning the third boy, 12 years old, who stayed in an almshouse together with his mother and sibs, remarks are made that girls have often complained about his »forwardness». Later, on a given occasion, he was guilty of a »shameful assault» on a girl of 15 years who was sleeping in the same room as himself and the rest of the family.

As to the 16 cases in Group III (more pronounced delinquency) in 2 cases the remarks simply said »acts offensive to deceny». Of the remaining boys, aged 12—14 hade 6 made repeated attempts at coitus with small girls between 3 and 5 years old. Another boy, 11 years old, who came from a good home was stated to have been »mixed up in an immorality affair, in which fornication was committed with small girls, including his own sister». — Two boys, 14 and 15 years old, had both been members of a gang that had sexual intercourse with girls. Concerning 2 other boys the record merely says that, besides several other offences,

they had been »mixed up in an immorality affair». In the remaining 3 cases it was a question of homosexuality. One of them, 17 years old, was given a conditional sentence for »fornication against nature». The remaining two boys were 14 years old. One of them had burnt a younger boy on the genitals and injured him to such an extent as to require hospitalization. Finally, one boy who had committed several thefts and often played truant, had also proved »a nuisance to his male playmates».

General delinquency is not very frequent among these boys. Only relatively few have been taken into public charge for this reason alone, though more often among the slight cases of delinquency than among the severe. General delinquency combined with other types is somewhat more common.

A question that may be of some interest is whether the boys have committed their offence or, rather, offences by themselves alone or in company with one or more fellows, or whether perhaps they have been members of gangs. On going through the material with a view to his point we find that a great majority of the boys ondoubtedly played their tricks single-handed — in so far as any remarks are put down in this respect — but over one-third (638 boys) have been in concert with school mates, whereas hardly one-tenth (134 boys) have been members of gangs. In the group of relatively slight delinquency (Group II) only 2 boys were members of a gang, while respectively 91 and 41 boys in the group of real delinquency (Group III) in the first and later period have preferred this kind of group formation. So, broadly speaking, nearly 1 out of every 5 delinquent boys has been interested in this kind of fellowship. That these boys have also operated single-handed may be taken for granted. Most of the gangs entered in this material have, of course, had crimes against property as their object. But gangs of another character are also represented. Thus 10 boys were mixed up in gangs which not only were thieving but were also devastating and doing a lot of damage. Another gang was mostly given to mischief and owdiness, while another devoted itself to vagabondage and begging. Several juveniles belonged to gangs that specialized in breaking slot machines and burglary of kiosks, while 4 boys belonged to a gang that was devoted to gross cruelty to animals. Also gangs

with sexual offences as their only purpose are represented. The gangs of the latter character included girls as members too — as was only to be expected. This is otherwise unusual. For the sake of completeness, it may be mentioned that the present material includes 6 girls in the first period and 3 girls in the later period who were members of gangs, mostly with a sexual purpose, but which also committed mischief and theft. No gang made up of girls alone has however been recorded.

As mentioned before, the essential features of the various types of delinquency encountered among the boys apply on the whole to the girls too. Only when it comes to sexual delinquency are the types different for the girls and boys — and this applies not only to delinquency but also to more criminal acts of a sexual nature. As pointed out already, sexual delinquency is less frequent among the boys than among the girls.

Sexual delinquency in girls hardly requires any special comment. Nor does it give rise to any diagnostic difficulties. This at least is not the case where the more common types are concerned, namely: cases in which it is plainly evident that the girls at too early an age or unrestrainedly and without discrimination, have abandoned themselves to sexual escapades with various persons for the sake of gain, i.e. what in earlier days was called prostitution. In such cases the difficulty has chiefly been to interpret the remarks put down by the informants. Expressions such as »running about», »fond of boys», »hanging out mostly in doorways and stairways» etc. — remarks suggesting that the individual has not been very scrupulous sexually — are not considered sufficient to place her in the group of sexual delinquency. For this group comprises only cases in which the girls themselves have admitted to offences of this kind, or in which definite proof of this has been furnished.

The general type »sexual delinquency» includes also offences of sexual nature where the co-operation of the girls themselves has been less pronounced as in cases of incest or utilization of small girls by older men. So we may expect a certain distribution of the cases according to the age of the girls as well as according to the various nature of the delinquency.

From Table 21 it will be noticed that certain differences assert

Table 21. **Distribution on different types of delinquency in girls.**[1]

Types of delinquency	1903—1925		1926—1940		1903—1940	
	No.	% of N	No.	% of N	No.	% of N
Group II						
Total number of girls = N	52	—	23	—	75	—
Crimes against property	15	28.8 ± 6.3	8	34.8 ± 9.9	23	30.7 ± 5.3
of these: Malicious damage	—	—	—	—	—	—
Vagrancy	29	55.8 ± 6.9	12	52.2 ± 10.4	41	54.7 ± 5.7
Sexual offences	1	1.9 ± 1.9	4	17.4 ± 7.9	5	6.7 ± 2.9
General delinquency	17	32.7 ± 6.5	3	13.0 ± 7.0	20	26.7 ± 5.1
Group III						
Total number of girls = N	87	—	160	—	247	—
Crimes against property	58	66.7 ± 5.1	48	30.0 ± 3.6	106	42.9 ± 3.1
of these: Malicious damage	6	6.9 ± 2.7	—	—	6	2.4 ± 1.0
Vagrancy	45	51.7 ± 5.4	86	53.8 ± 3.9	131	53.0 ± 3.2
Sexual offences	17	19.5 ± 4.3	91	56.9 ± 3.9	108	43.7 ± 3.2
General delinquency	15	17.2 ± 4.1	20	12.5 ± 2.6	35	14.2 ± 2.2

[1] Every case is counted as many times as it belongs to the different types.

themselves in the frequency of the types of delinquency among the *girls*, both between the various groups of slight and more severe cases and between the two periods.

Among the cases of minor delinquency (Group II) vagabondage is the most common type in both periods, and more than one-half of the girls have been given to this — the type of passive escapades. In the first period the next most common type is general delinquency, while crimes against property comes third. Sexual offences account for a very small percentage (2 per cent). In the later period, on the other hand, crimes against property become more frequent (35 per cent), and sexual offences likewise become more conspicuous (17.4 per cent), while general delinquency is lowest in frequency (13 per cent). The absolute figures in the later period are so small, however, that accidental variations cannot be excluded.

Among the more severe cases of delinquency (Group III) a

distinct difference asserts itself in the frequency of the various types of delinquency in the two periods. In the first period crimes against property occupy the first place (67 per cent), then comes vagabondage (52 per cent) while sexual offences and general delinquency are lowest (20 and 17 per cent respectively). In the later period the highest incidence is found for sexual delinquency (57 per cent) though insignificantly higher than that for vagabondage (54 per cent). That the difference between these groups is no greater might at least in part be due to the circumstance, as mentioned before, that only fully established instances of sexual delinquency are reckoned as belonging to the former group, whereas all doubtful cases of this type are entered in the latter group. So the present figures for sexual delinquency have to be looked upon as minimum figures. If, however, we also include the formally uncertain, though probably certain, cases, we have an additional 49 cases, and a total ratio of 87 per cent, which has to be looked upon as a maximum figure. The remarks in the records on which this calculation is based are, for instance, »running about», which is found in 30 cases. In the other cases the statements have been »fooling with boys», »running about in the streets at night», »camping with boys», etc. Most likely these figures are nearer the truth than the above minimum figures.

Further, a pronounced difference is found in the frequency of sexual delinquency between the two periods, with a higher frequency in the later period. This difference amounts to 37.4 ± 5.8 %, being thus statistically significant. Most likely this is due to the circumstance that in the first period the upper age limit for intervention by the Child-Welfare Board was 15 years — exceptionally a little higher — and consequently the most frequent type of delinquency among the girls would largely fall outside the authority of the board.

Table 22 shows how many girls were given to one or the other type of delinquency. Just as in the case of the boys, most of the girls among the slight cases of delinquency (Group II) have kept to one and the same type, mostly vagabondage, which occurred to about the same extent in both periods. In the first period general delinquency was somewhat more frequent, while

Table 22. **Distribution of delinquency in girls on different combinations of types.**

Types of delinquency	1903—1940			
	II		III	
	No.	%	No.	%
1. Crimes against property	11	14.7 ± 4.1	32	13.0 ± 2.1
2. Vagrancy	29	38.6 ± 5.6	31	12.6 ± 2.1
3. Sexual offences	5	6.7 ± 2.9	54	21.9 ± 2.6
4. General delinquency	16	21.3 ± 4.7	10	4.0 ± 1.3
5. Crimes against property + vagrancy	10	13.3 ± 3.9	48	19.4 ± 2.5
6. Crimes against property + sex. off.	—	—	10	4.0 ± 1.3
7. Crimes against property + gen. del.	2	2.7 ± 1.9	6	2.4 ± 1.0
8. Vagrancy + sexual offences	—	—	30	12.2 ± 2.1
9. Vagrancy + general delinquency	2	2.7 ± 1.9	9	3.6 ± 1.2
10. Sexual offences + general del.	—	—	4	1.6 ± 0.8
11. At least 1—4 in conjunction	—	—	13	5.3 ± 1.4
Total	75	100.0	247	100.0

in the later period the incidence of crimes against property was higher. Only a few girls in this group had committed sexual offences — in both periods altogether 5 girls. Owing to the dissimilarity pointed out above within the sexual type itself, it seems appropriate to give a more detailed account of these five cases.

In one case the offence consisted of incest. The girl was 14 years old. Her father was a labourer who had repeatedly had coitus with an older daughter also. The home was reported to be very poor, and the family received full municipal relief. The dwelling was dirty and cramped; the husband, wife and both daughters, respectively 14 and 16 years old, slept in the same bed.

Another case was that of a girl, 12 years old, who had been a member of a gang with a sexual purpose. — Two girls, respectively 11 and 7 years old, had repeatedly visited two older men who had utilized them sexually. Both girls came from decidedly bad homes, one was said to be of gypsy origin, and both were of illegitimate birth. — The youngest of the 5 girls was only a little over 6 years old and had been running about in the streets, going home with any man, and receiving »sweets» in return. The father, a gardener, had been gaoled and sentenced for theft and maltreatment of his wife. The parents were divorced, and the mother had to take care of 6 children. The home was poor, and the mother had sexual intercourse with a »lodger».

As to Group III, severe cases of delinquency, it will be noticed that in the first period a majority of the girls (58 out of 87) had been guilty of crimes against property alone or combined with vagabondage or, in a few cases, with general delinquency. Here sexual offences are less conspicuous, and only 17 girls are recorded for this type either alone or in connection with other offences. Among these girls also are found differences in the nature of the delinquency (cf. Table 21).

The three youngest of these girls, between 8 and 11 years, had been utilized by older men. In one case, a girl, aged 11, had played the rôle of leader and tempter, enticing other smaller girls to repeated visits to an elderly debilitated man, with whom she had carried on as she pleased. After such visits she had played the rôle of »benefactor» and invited the other girls to street-car rides and cakes. The girl, who was brought up by her paternal grandparents, as first her father and, later, her mother had left the home, had been poorly looked after.

It looks indeed as if sexual delinquency of this kind is not so much to be blamed on the girls themselves as on unfortunate extraneous circumstances. But girls are far from always being so innocent as they may appear. Often, as illustrated by the case just cited, they know very well how to use a given situation to their own advantage.

Of the remaining 3 girls, between 9 and 12 years old, the youngest had been a member of a gang with a sexual purpose. In spite of the young age — just 12 years — the 2 other girls had shown sexual delinquency of the usual type, and thus both were presumably cases of hypersexuality. In the remaining 11 cases all the girls were over 14, on an average 14.9 years. Here it generally is a question of sexual delinquency of common character, i.e. staying with various men for gain. Six of the girls had been picked up by the police while roaming about in the streets for immoral purposes, and 2 had been staying on boats in the harbor. They could all be considered as definite cases of prostitution.

In the later period more than one-half of the girls (91 out of 160) were taken in charge on account of sexual misconduct alone or in combination with some other type of delinquency. As in the preceding cases, the younger girls had chiefly been members of sexual gangs or stayed with older men, while the older girls had turned to sexual delinquency of the usual type.

On investigation into the occurrence of combinations of the various types of delinquency, it was found that more than one

type was relatively seldom represented simultaneously among the slight cases of delinquency (group II). The most common combination is crimes against property and vagabondage — in reality, petty larceny combined with truancy — which is encountered in about 13 per cent of the cases. Among the more severe cases of delinquency (group III) it is noticeable that the types are kept »pure» in only a little more than half of the cases. Compared with the former group (II) a significant difference thus exists (29.9 ± 5.5). Among those committing two or more types of delinquency the combination of vagabondage with crimes against property is the most common in the first period, being encountered in a little over one-fourth of the cases. In the later period, however, the combination of sexual delinquency with vagabondage is more frequent, though only insignificantly so, than the aforementioned combination.

In the first period there is a certain parallelism between the frequency of the types of delinquency among the boys and girls. In Group II vagabondage is most common in both sexes, followed by crimes against property and general delinquency, whereas sexual delinquency is represented only by very low percentages in both sexes.

Also in Group III there is some degree of parallelism in this respect between the boys and girls in the first period, crimes against property being the most frequent, then vagabondage. Still, it is noticeable that the girls commit crimes against property to a lesser extent than the boys. The difference between the types of delinquency among the boys and girls does not manifest itself until the later period, when sexual delinquency becomes far more frequent among the girls than among the boys. In the first period this difference amounts to 17.5 ± 4.3 %, while it is far more pronounced in the later period, being here 53.4 ± 4.1 %, and thus statistically significant in both periods.

Presumably this difference is due to difference in the age limit between the later and the first periods. The girls have attained the age of puberty, on which account the sexual offences become more common among them. In boys the stage of puberty is later in its appearance. Furthermore, sexual offences

are judged less severely in the case of boys. Most likely, this is a contributary cause of the difference. It is a striking fact, however, that in this period crimes against property become less frequent among girls than among boys. Most likely, this is due to the fact that girls are more cautious and timid than boys — rather than to a stronger acknowledgement among girls of the sacredness of property.

Summarizing the present findings, then, it is evident that in the first period the delinquency of both sexes manifested itself similarly. Vagabondage is predominant among the slight cases of delinquency, while crimes against property are most frequent among the severe cases. In the later period a dissimilarity in tendency and temperament appears between the boys and the girls, the girls being more given to sexual escapades, while the boys stick to more concrete things in the form of crimes against property. Most likely, however, boys commit sexual offences just as often as girls. But as puberty appears later in boys than in girls, misconduct of this kind does not occur among boys to any great extent till a later juncture, when such conduct is looked upon as rather natural and is not treated as delinquency.

The differences between the earlier and the later period should therefore depend on the alteration in the age limit between the two series.

No real difficulty has been encountered in classifying the court cases as they have been classified according to the type of offence recorded in the verdict. Where more than one type of offence is recorded they have, as can be seen, been classified accordingly.

The position can be seen from the table and comment is scarcely necessary. It is well known, and is confirmed by the table, that in so far as crimes against property are concerned, girls are usually guilty of petty larceny whereas boys engage in the more serious forms of such crime. It is also apparent that 14 out of 692 boys (2.0 per cent) and 3 out of 53 girls (5.7 per cent) were guilty of sexual offences. Relatively considered therefore, sexual crimes are also commoner among women than among men, just as we have seen previously among cases of

Table 23. **The sentenced cases (group IV) percentually distributed according to cause of sentence.**

Cause of sentence	Boys		Girls	
	1903—1925	1926—1940	1903—1925	1926—1940
Petty larceny	26.2	22.8	46.6	39.1
Theft	34.1	18.7	40.0	21.8
Burglary	29.3	31.1	6.7	—
Forgery, fraud and embezzlement	7.7	7.6	6.7	21.8
Sexual offences	1.5	2.4	—	13.0
Illicit car-borrowing	—	7.1	—	—
Illicit car-borrowing + theft	—	7.3	—	4.3
Assault and battery + malicous damage	1.2	2.7	—	—
Running away		0.3	—	—
Total	100.0	100.0	100.0	100.0
No. of cases examined	324	368	30	23

delinquency where sexual offences are commoner among girls than among boys.

On closer examination it is found that two of these girls were guilty of offences against the Venereal Disease Law and with the third it was a case of criminal abortion. Among the boys there were 4 cases of homosexuality, 8 of coitus with girls under age and 2 of attempted rape.

No great difference would be expected between those actions which led to intervention by the Child-Welfare Board and those which led to legal proceedings. However, where the girls are concerned, as a large number of their offences were of a sexual nature, a certain difference can be seen. This is because the same types of offence which in the younger age-groups lead to intervention by the Child-Welfare Board, come, in the older age-groups under the Law against Vagrancy and lead to a caution against vagrancy or sentence to forcedlabour. This however cannot happen at those ages with which the present investigation is concerned.

CHAPTER VI.

HOME CONDITIONS DURING CHILDHOOD.

If we are to understand the problem of delinquency, it is not enough to expound the various manifestations of delinquency. It is equally important to investigate the conditions under which the delinquent children grow up — primarily their homes. Previous investigations give the general impression that a great majority of the delinquent children came from »bad» homes. It is therefore to be regretted that no comparative material as to the domestic aspects is available for non-delinquent children. Naturally a certain percentage of the latter must also come from bad homes, but we do not know how high this percentage is. Thus we have to limit ourselves to a comparison of the various groups in the material.

In order to get a general idea about the conditions during growth an investigation was first made into the extent to which these children enjoyed the care of both parents in early childhood, or when this was not the case, with whom they lived, and, further, whether their situation had undergone any change at the juncture when the Child-Welfare Board took charge of them.

It is obvious that Group I occupies a special position, and that this to some extent applies also to Group II, as here we are dealing with children who were taken in charge entirely or partly on account of bad conditions at home. As will be noticed, in the first period nearly 80 % of the boys in both groups spent their first years with both parents, and on the whole the same applies to the later period (cf. Table 24).

At the point of time for the intervention by the Board the situation was different, as in the first period only a little over

Table 24. **The boys percentually distributed according to whom they were living with before and when taken care of or sentenced.**

Living with	Group I		Group II		Group III		Group IV	
	When first observed	When taken care of	When first observed	When taken care of	When first observed	When taken care of	When first observed	When taken care of
				1903—1925				
Both parents	79.1	27.1	78.9	39.4	88.9	55.4	94.2	69.6
One of the parents	17.5	69.5	11.5	55.8	4.9	35.5	3.7	23.5
Near relatives	1.7	1.1	5.8	2.9	2.1	4.8	0.9	2.5
In foster home or children's home	1.7	2.3	3.8	1.9	4.1	4.3	1.2	2.2
In service (own lodgings)	—	—	—	—	—	—	—	2.2
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	177	177	104	104	386	394	324	323
				1926—1940				
Both parents	74.6	44.1	79.4	32.4	80.6	39.3	86.9	66.6
One of the parents	20.9	53.0	10.3	55.8	7.4	40.6	3.8	24.0
Near relatives	1.5	—	7.4	4.4	3.2	5.5	4.4	4.4
In foster home or children's home	3.0	2.9	2.9	5.9	8.8	12.3	4.9	3.9
In service (own lodgings)	—	—	—	1.5	—	2.3	—	1.1
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	67	68	61	61	217	219	366	362

one-fourth of all the boys in Group I and less than one-half of the boys in Group II lived with both parents. In the later period nearly one-half (44 %) and one-third (32 %) of the boys in the two respective groups lived at home with both parents. It may be that in the first period more stress was laid upon homes characterized as incomplete or broken up, so that expecially children born out of wedlock and children whose homes were broken up by death of the parents became subject to public care; and thus a certain selection of the material took place. In the later period other means have been available for the aid of mothers having to fend for themselves, without taking care of their children, so that the relative number of such children taken in charge by the community should naturally be reduced.

Of the boys in Groups III and IV about 90 % had spent their first years of life together with both parents. This applies to the first period. In the second period the number of such boys was not quite so high in either group. At the time of intervention by the Child-Welfare Board or the Court, a change had taken place in this respect — just as in the preceding groups — as less than one-half of the delinquent boys in Group III and two-thirds of the criminal juveniles (Group IV) were then livning with both parents.

As regards parental supervision, then, the criminal boys were better off than the delinquent ones. The difference between these two groups is statistically significant both for their earlier childhood and at the time of intervention by the Board or Court; and this holds good for both periods.

As to the girls, in Groups I and II the situation in early childhood was about the same as that of the boys in the corresponding groups. (Cf. Table 25.) At the time of the Board's intervention the situation had changed for the girls, too. Thus, in the earlier period only one-fifth and one-fourth of the girls in the two groups respectively were under the care of both parents. In the later period — just as was found for the boys — a greater part of the girls, nearly one-half of them were living with both parents. This can probably be explained in the same way as for the boys. In Group II, on the other hand, hardly one-fifth of the girls were living with their parents at the same point of time, making a lower percentage than that of the boys in the corresponding group.

Of the delinquent girls (Group III) three-fourths (75.9 %) spent their first years together with both parents in the earlier period, two-thirds (66 %) in the later period. At the time of intervention by the Board in the first period less than one-half (41.5 %) were under the care of both parents, in the latter period only one-fourth (25.6 %).

Comparison with the boys in the same Group (III) shows the percentage of children growing up under the care of both parents to be lower for the girls than for the boys in both periods. The difference for both periods respectively amounts to 13.0 ± 3.2 % and 14.6 ± 4.6 %, and is thus statistically signi-

Table 25. **The girls percentually distributed according to whom they were living with before and when taken care of or sentenced.**

Living with	Group I		Group II		Group III		Group IV	
	When first observed	When taken care of	When first observed	When taken care of	When first observed	When taken care of	When first observed	When taken
1903—1925								
Both parents	78.3	20.8	80.4	26.9	75.9	41.5	90.0	33.3
One of the parents	15.4	69.6	9.8	61.6	6.9	40.2	6.7	36.7
Near relatives	2.3	3.4	2.0	1.9	4.6	4.6	—	—
Foster home or children's home	4.0	6.2	7.8	9.6	12.6	12.6	3.3	3.3
In service (own lodgings)	—	—	—	—	—	1.1	—	26.7
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	175	178	51	52	87	87	30	30
1926—1940								
Both parents	75.8	49.2	60.9	17.4	66.0	25.6	81.9	31.8
One of the parents	22.7	49.2	30.4	60.9	17.3	38.1	13.6	40.9
Near relatives	1.5	—	—	—	7.7	7.5	4.5	—
Foster home or children's home	—	1.6	8.7	21.7	9.0	17.5	—	—
In service	—	—	—	—	—	11.3	—	27.3
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	66	65	23	23	156	160	22	22

ficant. At the time of intervention by the Board the girls were also worse off as regards parental supervision than were the boys, and the difference is significant here too.

As regards spending their childhood together with both parents, the criminal girls (Group IV) were largely in the same position as the boys in this group. But at the time of intervention by the Board the conditions appear to have been less favourable for these girls than for the corresponding boys, but the number is too small to allow of any statistical comparison.

It may be of interest to look into the childhood of the children who from the very start had to do without the care of either parents. In Groups I and II most of these children lived with one of the parents, which as a rule means the mother. Only in one instance did the father look after the child from the very

beginning. A small number of children were left in the care of relatives, which most often means the maternal or paternal grand-parents, mostly the former, and in a few cases in the care of maternal uncles or aunts, or other relatives. The number of children who have spent the first year of life with their mother is relatively greater in Group I than in the other groups, presumably because, as will be demonstrated later, children born out of wedlock are more numerous in this group. So, of course, in most of these cases the child stayed with the mother. Furthermore, illegitimacy is often associated with bad home conditions, and indeed this circumstance has lain behind intervention by the Board in this group. Of the other children belonging to Groups I and II, a relatively small number of boys grew up in asylums in the earlier period and the somewhat greater number in the later period, while the reverse applies to the girls. Children with a pronounced tendency to delinquency (Group III) who are not living with both parents, appear to have spent their early childhood just as often with the mother as in asylums. In the latter period, however, a relatively greater number of girls grew up under their mother's care than in the earlier period. The difference amounts to 10.4 ± 4.1 % and is thus statistically probable.

Several of the children who from the start have been under the care of others than their parents have also experienced a change of environment. Consequently at the time of intervention by the Board a certain percentage of these children have lived in different surroundings. The varying sojourns of these children after the change in milieu is really of minor interest. The often reiterated statement that children left to the care of the grandparents run a greater risk of becoming delinquents appears not to be corroberated by the present material, insofar as neither the child-welfare clientele proper nor the juvenile court clientele includes any strikingly high percentage of children who were looked after by their grandparents.

Table 26. **Mortality in percent during the first year of life of legitimate and illegitimate children in Schweden.**

(Swedish Official Statistics.)

Annually	Legitimate	Illegitimate
1901—1910.....	7.8	13.2
1911—1920.....	6.4	10.2
1921—1930.....	5.6	7.6
1931—1935.....	4.7	6.5
1936—1940.....	3.9	5.9

ILLEGITIMATE BIRTH.

In earlier times in particular, children born out of wedlock were badly situated. They were looked upon as inferior, and treated accordingly. The mortality among them was much higher than among the legitimate children, as is evident from Table 26.

Several factors contributed to this unfortunate state. One was of financial nature. It is obvious that an unmarried and solitary mother has a hard time both to provide for a child and to give it an adequate education. Moreover — and this is particularly true of earlier days — such mothers were often liable to social ostracism. Naturally under such circumstances, most of the women who ran the risk of illegitimate children were a little thoughtless or lax, or they were women who fell in with men of similar character. The times have changed, however. Social ostracism is no longer so severe as formerly and society supports unmarried mothers, both financially and by helping them to take care of their children properly. Undoubtedly these circumstances have helped to reduce the difference in the mortality between children born in and out of wedlock.

The normal figures in Table 28 are calculated from official statistics. Figures available for the town of Malmö refer to the natives of this town. For the »immigrants» figures have been used that apply to the rest of the population of this county. These figures may be a little too high for the immigrants, most

Table 27. **Number of illegitimate children in the different groups.**

Group	1903—1925		1926—1940		1903—1940	
	No. of cases	of these illegi-timate	No. of cases	of these illegi-timate	No. of cases	of these illegi-timate
			Boys			
I	177	56	69	23	246	79
II	105	35	61	18	166	53
III	396	82	227	60	623	142
IV	324	27	368	63	692	90
Total	1 002	200	725	164	1 727	364
			Girls			
I	178	56	66	23	244	79
II	52	12	23	7	75	19
III	87	30	160	66	247	96
IV	30	3	23	4	53	7
Total	347	101	272	100	619	201

of whom come from the villages. The frequency of illegitimate children is greater in the towns than in the villages. So we may consider the normal figures are on the high side for the respective periods. It should also be pointed out that the figures apply throughout to the conditions at birth. Several of the children born out of wedlock are later legitimized by the marriage of their parents, so that rather over one-half of the children born out of wedlock are said to be legitimized within their first 5 years of life (HYRENIUS, HOLMSTEDT). This has not been taken into consideration in the calculation of the normal figures, nor in the figures for my material, and they are therefore quite comparable, since relevant data in the present material have been obtained first from the records of the Child-Welfare Board and have later, in most cases, been checked up from information obtained directly from the parish registers and corrected accordingly.

As is evident from Table 28, the number of illegitimate children in the material as a whole and for both periods in one, makes a relatively high percentage, namely: 21.1 % for

Table 28. **Observed percentage number of illegitimate children among the cases and calculated percentage number of such children according to Swedish Official Statistics. (Malmöhus county.)**

Group	Boys			Girls		
	Observed number illegitimate children %	Calculated number illegitimate children %	Difference, %	Observed number illegitimate children %	Calculated number illegitimate children %	Difference, %
			1903—1925			
I	31.6	18.7	+ 12.9 ± 2.9	31.5	18.4	+ 13.1 ± 2.9
II.....	33.3	17.4	+ 15.9 ± 3.7	23.1	18.1	+ 5.0 ± 5.3
III	20.7	17.7	+ 3.0 ± 1.9	34.5	17.8	+ 16.7 ± 4.1
IV	8.3	16.4	− 8.1 ± 2.1	10.0	15.7	− 5.7 ± 6.6
Total	20.0	17.1	+ 2.9 ± 1.2	29.1	17.8	+ 11.3 ± 2.0
			1926—1940			
I	33.3	19.9	+ 13.4 ± 4.8	34.8	19.5	+ 15.3 ± 4.9
II.....	29.5	19.7	+ 9.8 ± 5.1	30.4	20.9	+ 9.5 ± 8.5
III	26.4	20.4	+ 6.0 ± 2.7	41.3	19.5	+ 21.8 ± 3.1
IV	17.1	19.1	− 2.0 ± 2.0	17.4	19.6	− 2.2 ± 8.3
Total	22.6	19.6	+ 3.0 ± 1.5	36.8	19.6	+ 17.2 ± 2.4
			1903—1940			
I	32.1	19.0	+ 13.1 ± 2.5	32.4	18.7	+ 13.7 ± 2.5
II.....	31.9	17.6	+ 14.3 ± 3.0	25.3	20.0	+ 5.3 ± 4.4
III	22.8	17.7	+ 5.1 ± 1.7	38.9	20.1	+ 18.8 ± 2.0
IV	13.0	17.8	− 4.8 ± 1.5	13.2	17.4	− 4.2 ± 5.2
Total	21.1	18.4	+ 2.7 ± 0.9	32.5	18.7	+ 13.8 ± 1.6

the boys and 32.5 % for the girls, as against 18.4 % and 18.7 % respectively for boys and girls in the normal population. For the boys the difference amounts to 2.7 ±0.9 % and for the girls 13.8±1.6 %; that is, the difference for boys and girls alike is statistically significant.

In the present material, however, the high total figures may be due to Groups I and II, comprising children taken in charge by the Board entirely or partly on account of bad home conditions. As is evident from Table 27 nearly one-third of these children were born out of wedlock, and this applies equally to boys and girls, and nearly to the same extent in both periods.

It is obvious that the risk of being taken charge of by the board is almost twice as great for the illegitimate children as for those born in wedlock. This will justify the conclusion that there is a close connection between bad home conditions and illegitimate birth, in other words the same mentality that leads to the birth of children outside the pale of society may also lead to deterioration in the home. It should be kept in mind, however, that, according to the legal regulations since 1918 the homes in which the illegitimate children are cared for have been under the supervision of the Child-Welfare Board — a circumstance that does not apply to the homes of children born in wedlock. Consequently, it may be that mismanagement of the former homes is observed more often and thus brought before the Board to a greater extent than similar neglect of the latter homes, so that both Groups (I and II) become somewhat selected.

For boys from the delinquency clientele proper (Group III) the ratio of illegitimate children in the first period is only a little higher than the normal figures from the same period. (Cf. Table 28.) The difference, 3.0 ± 1.9 %, is not significant and may be due to random variation. For the later period the difference is greater, amounting to 6.0 ± 2.7 %, but this is not significant, either. In the present material, then, the illegitimate boys are no more numerous in the delinquency contingent than what corresponds to their relative part in the population.

In Group IV, criminal juveniles, we meet with the surprising result that the figures for the illegitimate children are very low. Evidently illegitimate birth does not play any great rôle in the age-groups concerned here.

For the period of 1903—1925 we even find the indicidence of illegitimate birth to be slightly lower among the criminal boys than in the normal population, the difference amounting to 8.1 ± 2.1 %. It is of course, inconceivable that illegitimate birth is an advantage involving a lesser risk of criminality. The opposite would rather be expected. The difference may to some extent be ascribed to the circumstance that, as mentioned before, the normal figures might be somewhat too high. But this can hardly be the whole explanation.

It should be kept in mind that AHNSJÖ (1941) arrived at the same result. He was able to show that in 1914—1918 the risk of being sent to a reformatory decreased for girls born out of wedlock, and increased for legitimate girls. In this respect the criminal juveniles in the present material are quite comparable to AHNSJÖ's material, and the results obtained are concordant. My material is too small, however, to permit of any further division in the years mentioned.

As a matter of fact, apart from this period, the figures show merely random deviations from the normal figures. From the above it is also quite evident that, on comparison between the criminal boys and the delinquent, the incidence of illegitimate birth is considerably lower for the former than for the latter; and this difference is significant for either period.

As mentioned in the introduction, the number of girls born out of wedlock is considerably greater than the corresponding number of boys. The difference for the entire material is 11.4 ± 2.12 %, *i.e.*, statistically proved. This feature is particularly pronounced in Group III — that is, the group of severe delinquency — where in the first period one-third of the girls (34.5 %) and in the later period nearly one-half of the girls (41.3 %) had been born out of wedlock. The difference from the frequency for the normal material is significant for both periods. On comparing the boys and girls in this group (III) it is likewise noticeable that in both periods the delinquent girls were illegitimate far more often than were the boys. Here, too, the difference between boys and girls is statistically significant.

We can probably rule out the possibility that birth out of wedlock should prove more detrimental for the girls than for the boys, so that the former would be more liable to detention. It is conceivable, however, that we have here a parallel to Groups I and II, where the homes are markedly bad, and where, moreover the frequency of illegitimate birth is particularly high; this may also have been the case with the delinquent girls, and they may more often than the boys come from particularly bad homes.

BROKEN HOMES.

Children born in wedlock, however, may also be placed in the same situation during growth as children born out of wedlock by the change that American authors have called »broken homes», *i.e.,* that the parental home is broken up by divorce or by the death of one of the mates.

Naturally the period in the life of the child at which the home is broken up is not without importance. It is only reasonable to assume that this means more to rather older children than to the quite little ones. It would have been interesting, therefore, to investigate at what time the home was broken up, and how long a period passed before the child was taken in charge by the Board, or showed the first symptoms of delinquency. But the data required for this purpose have not been entered in the records, and we must therefore content ourselves with investigating the frequency of broken homes.

The percentage of broken homes has been calculated in relation to the total number of children and to the number of children from homes where a split was possible, *i.e.,* children of married parents and those of unmarried parents living together. Illegitimate children whose parents were not living together cannot be included in this calculation because their homes have never been complete.

Table 29 shows that the highest frequency of broken homes is found in Groups I and II, i.e., for the children who were under the care of the Board directly or indirectly on account of unfortunate home conditions. This may partly be because these children constitute a selected material. Thus in the first period, in Group I over one-half of the total number of homes, and two-thirds of the potentially »breakable» homes had been dissolved at the time when the respective children were placed under the care of the Board. In the later period the corresponding figures are lower — which might be due to the same thing as mentioned before: change in administrative practice. The figures are lower for Group II than for Group I in the earlier period, but higher in the later period.

In the delinquency clientele proper (Group III) the boys

Table 29. **Percentages of children from »broken homes».**

Group	A	B	C	Percentage number of children from »broken» homes	
				C in per cent of A	C in per cent of B
Boys 1903—1925					
I	177	141	93	52.5 ± 3.8	66.0 ± 4.0
II	105	83	41	39.0 ± 4.8	49.4 ± 5.5
III	396	344	134	33.8 ± 2.4	39.0 ± 2.6
IV	324	303	83	25.6 ± 2.4	27.4 ± 2.6
Boys 1926—1940					
I	69	52	25	36.2 ± 5.8	48.1 ± 6.9
II	61	54	32	52.5 ± 6.4	59.3 ± 6.7
III	227	176	88	38.8 ± 3.2	50.0 ± 3.8
IV	368	324	78	21.2 ± 2.1	24.1 ± 2.4
Girls 1903—1925					
I	178	137	100	56.2 ± 3.7	73.0 ± 3.8
II	52	41	27	51.9 ± 6.9	65.9 ± 7.4
III	87	66	33	37.9 ± 5.2	50.0 ± 6.2
IV	30	27	12	40.0 ± 8.9	44.4 ± 9.6
Girls 1926—1940					
I	66	50	18	27.3 ± 5.5	36.0 ± 6.8
II	23	16	12	52.2 ± 10.4	75.0 ± 10.8
III	160	103	56	35.0 ± 3.8	54.4 ± 4.9
IV	23	19	8	34.8 ± 9.9	42.1 ± 11.3

A = Total number of homes.

B = Number of children where the parents were married or co-habitants at the birth of the child.

C = Number of homes at the time of intervention of the Board or the Court where the parents were not living together because of death or separation.

come from broken homes in a little over one-third of the possible cases in the earlier period, and in one-half of the possible cases in the later period. The difference between the two periods is significant for Group II as well as for Group III, and the increased frequency of broken homes in the later period may very likely be due to the increased risks of divorce in more recent years (DAHLBERG 1946).

The criminal boys (Group IV) again are better situated with regard to home conditions of this kind, as a smaller number of them come from broken homes. Still, broken homes are recorded in this group in one-fourth of all the cases in the earlier period, but only in one-fifth of the cases in the later period. It is rather difficult to explain why the increase in the incidence of divorce should not play a part in this group, however.

The difference between the two groups of boys, delinquent and criminal, in these home conditions, calculated on potential cases, amounts to 11.6 ± 3.7 % for the earlier period, 25.9 ± 4.5 % for the later period, and is thus significant. This difference indicates that the home conditions affect criminal boys less than delinquent boys. In other words, at the age attained by the criminal boys, the state of the home is of less consequence. The condition of the home plays a greater part where the delinquent boys are concerned, even though not constituting the only reason for intervention by the Board.

We find the same conditions in the earlier period for the girls as for the boys: the highest frequency of broken homes is found for Groups I and II, the lowest for Group IV. In the later period the frequency of broken homes is lowest for Group I, but — as mentioned above, for the boys — this is probably due to the changes in the rules of the Board. It is of particular interest that the figures are fairly consistently higher for the girls than for the boys, and this becomes particularly conspicuous on calculation of the frequency of the total numbers of boys and girls. In the earlier period 63 % of the girls came from broken homes as against 40 % of the boys. In the later period the homes had been dissolved for 50 % of the girls and only for 37 % for the boys. The difference for the two periods amounts to 23.1 ± 3.4 % and 13.2 ± 4.1 % respectively, thus being significant. The same is found on comparison between boys and girls in the various groups, except in Group I during the later period, and this later divergence may be due to chance. Accordingly the outcome of these investigations concerning broken homes is in keeping with what we have

repeatedly ascertained before: that delinquent girls live in a more unfavourable milieu than do delinquent boys.

Naturally, it is difficult to obtain comparative figures for the frequency of broken homes among the normal population. But it is hardly likely that the percentage of broken homes should be quite as high in the total population as in this material. In Malmö, during the period 1910—1940, the rate of widows and divorced women in percent of the number of married and formely married women was found to be 14—15 %. In the younger age-groups of course, the figures are lower. Although these figures are not directly comparable with the figures from the present material, they may still give a certain guidance in the assessment.

If, as claimed by American authors, broken homes are an important factor in delinquency, it is of interest to investigate why these homes were broken up. For it is conceivable that the cause may in itself influence the development of the child just as much as the actual dissolution of the home.

A survey of Table 30 shows that in practically all the groups and in both periods the death of one of the parents caused the breaking up of the home in most of the cases. It is noteworthy, however, that in the later period the difference between death and divorce is not so pronounced as before. The latter factor, divorce, is used in its broader sense, as including the cases where one of the parents has absconded without an official divorce. The absolute figures for this period are so small, however, that they allow of no general judgement.

It is further of importance to investigate which of the parents was most often missing in the home.

In Group I, where bad homes have prompted intervention by the Board, the mother had died in nearly twice as many cases as the father, and in the first period, refering to the boys, three times as often. If we also include the cases in which one of the parents absconded, the difference is equally pronounced. The same applies to Group II in which bad home conditions are also the rule. An exception is found, however, for the boys in this group during the first period, as here the father was missing, in the home more often than the mother. It is obvious,

Table 30. **Children from »broken» homes, percentually distributed according to the cause of the breaking up of the home.**

Sex and period	Cause of the breaking up of the home	I %	II %	III %	IV %
Boys 1903 —1925	Father dead	19 ± 4.1	32 ± 7.3	31 ± 4.0	49 ± 5.5
	Mother dead	60 ± 5.1	17 ± 5.9	28 ± 3.9	23 ± 4.6
	Both parents dead..	—	2 ± 2.2	1 ± 0.9	2 ± 1.5
	Father absconded...	8 ± 2.8	32 ± 7.3	22 ± 3.6	19 ± 4.3
	Mother absconded..	8 ± 2.8	5 ± 3.4	4 ± 1.7	—
	Parents divorced ...	5 ± 2.3	12 ± 5.1	13 ± 2.9	6 ± 2.6
	Total	100	100	99	99
	No. of cases	93	41	134	83
Boys 1926 —1940	Father deed	24 ± 8.5	22 ± 7.3	25 ± 4.6	29 ± 5.1
	Mother dead	32 ± 9.3	22 ± 7.3	31 ± 4.9	28 ± 5.1
	Both parents dead..	—	—	—	4 ± 2.2
	Father absconded...	16 ± 7.3	3 ± 3.0	7 ± 2.7	5 ± 2.5
	Mother absconded..	12 ± 6.5	9 ± 5.1	3 ± 1.8	—
	Parents divorced ...	16 ± 7.3	44 ± 8.8	34 ± 5.1	33 ± 5.3
	Total	100	100	100	99
	No. of cases	25	32	88	78
Girls 1903 —1925	Father dead	19 ± 3.9	11 ± 6.0	45 ± 8.7	33
	Mother dead	46 ± 5.0	41 ± 9.5	21 ± 7.1	25
	Both parents dead..	—	33 ± 9.1	6 ± 4.1	—
	Father absconded...	16 ± 3.7	—	15 ± 6.2	25
	Mother absconded..	9 ± 2.9	4 ± 3.8	—	8
	Parents divorced ...	10 ± 3.0	11 ± 6.0	12 ± 5.7	8
	Total	100	100	99	99
	No. of cases	100	27	33	12
Girls 1926 —1940	Father dead	17	17	25 ± 5.8	25
	Mother dead	33	25	36 ± 6.4	25
	Both parents dead..	—	—	5 ± 2.9	—
	Father absconded...	6	17	16 ± 4.9	25
	Mother absconded..	17	17	2 ± 1.9	—
	Parents divorced ...	28	25	16 ± 4.9	25
	Total	101	101	100	100
	No. of cases	18	12	56	8

however, that the mother plays a greater part than the father as being more directly responsible for the general character of the home. To some extent this is only natural. To the younger children the death of the father is less of a disaster than the death of the mother. The decease of the father is of financial consequence, but the decease of the mother influences more directly the care and management of the children.

As regards the homes where the children themselves have gone astray (Groups III and IV), the absence of the father plainly plays a more decisive part. Both the delinquent and the criminal girls have in both periods lost their fathers more often than their mothers. The same applies to the criminal boys, and also to the delinquent boys. In the later period, however, the delinquent boys have lost their mothers more often than their fathers. The difference however is not significant, and, in view of the large standard error has to be looked upon as uncertain. In the other cases the difference is proved.

COHABITANT PARENTS.

Just as legitimate birth does not always imply that the children will be reared by both parents, illegitimate birth does not always mean the opposite. Children born out of wedlock also may be able to profit by the care and supervision of both parents. As a matter of fact, several illegitimate children come from permanent relationships of matrimonial character, »non-registered marriage», usually known as cohabitant parents. It is found that 6.5 per cent of the children in the total material come from unions of this kind, and further that 27.1 % — that is over one-fourth — of the illegitimate children have, or have had, cohabitant parents. On the whole the frequency of such parents is the same for the various groups of the material though, naturally, somewhat higher for Group I. Furthermore, the frequency of cohabitant parents is higher for the boys than for the girls, and higher for the earlier period than for the later.

CHANGE OF HOME.

Not only the character of the conditions under which the children grow up is of importance to their development, but also the changes taken place in their surroundings. Accordingly the material has also been analysed with regard to such changes — by which is meant the transference of a child into an entirely new home, that is into unfamiliar surroundings.

Change of environment has occurred somewhat more often in the later period than in the earlier, and more frequently for the girls, the figures being respectively 8.3 per cent and 11.5 per cent for the boys, 13.6 per cent and 28.9 per cent for the girls.

During the period of 1903—1925 there was no great difference between the various groups with regard to a change of environment. During the period of 1926—1940, however, a change in milieu appears to have been more frequent among the Child-Welfare clientele than in the remainder of the material. This applies in particular to the more qualified cases of delinquency among the girls. Considering Groups II and III together, as compared to Group IV, we find that no less than 10.9 per cent of the delinquent boys have changed their environment at least twice, while the corresponding figures for the criminal boys (Group IV) are only 3.0 per cent — *i.e.*, a significant difference.

Similar conditions are found for the girls. No less than 11.7 per cent of the delinquent girls (Groups II and III) have changed environment at least twice, whereas the corresponding figures for the criminal girls (Group IV) is 2.3 per cent. So the difference is significant here.

SOCIAL POSITION OF THE FAMILY.

In order to get an idea of the social milieu from which these children come, it is of interest to study which social class they belong to. The material is therefore divided into four categories according to the ocupation of the fathers or mothers. For the first three social classes the basis of classification has been the same as in the official election statistics. Class IV has

been added by the author, covering, as will be noticed, persons with rather uncertain occupations. For the sake of easy survey, the occupations to be reckoned with for the urban population are entered in the various groups as follows:

Class I: Officers, higher officials, manufacturers, wholesale dealers and academic professions.

Class II: Officers in merchant vessels, shopkeepers, elementary-school teachers, artisans, clerks, foremen and minor officials.

Class III: Labourers, seamen, domestic servants, waitresses, rural workers.

Class IV: Persons without steady work — as »horse-traders», »wire-workers», »Chronically unemployed» etc.

As is evident from the review of the literature, delinquent children have been previously found not to come in any marked degree from the higher classes of society. This may be because the upper classes have a chance of getting their unsuccessful children cared for privately, in homes or institutions, and thus protect them from intervention by the authorities. The latter possibility appears at any rate to be able to play a certain part, but of course this varies in the various countries.

A distribution of the material on social principles is given in Tables 31 and 32. To see whether any one social class is represented more often here, the material must be compared with the total population. For this purpose I have made use of the afore mentioned official election statistics for 1921—1940 for the towns of Malmö, Hälsingborg, Landskrona and Lund, which are all in the same constituency (cf. Table 32). It may be mentioned that the relative figures for the normal population are averages for the afore mentioned election years, and that only three classes are used officially. For the sake of comparison, therefore, my social classes III and IV have been made into one and furthermore given only for the years 1926—40.

It will be noticed that the parents of the children in this material more often come from the lower classes of society than in the normal population. There is a statistically significant difference between the percentual number of men be-

Table 31. **Percentage distribution of the cases on different social classes.**

Social class [1]	Boys			Girls		
	1903—1925	1926—1940	1903—1940	1903—1925	1926—1940	1903—1940
			Group I			
I	—	—	—	—	—	—
II	3.8	5.8	4.4	8.1	6.2	7.7
III	89.3	65.4	82.5	89.7	66.7	83.6
IV	6.9	28.8	13.1	2.2	27.1	8.7
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	131	52	183	135	48	183
			Group II			
I	1.1	—	0.7	—	—	—
II	4.3	16.7	8.9	2.4	5.3	3.3
III	82.6	75.9	80.1	85.4	89.4	86.7
IV	12.0	7.4	10.3	12.2	5.3	10.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	92	54	146	41	19	60
			Group III			
I	0.3	2.4	1.0	—	—	—
II	12.1	13.3	12.6	2.7	12.6	9.0
III	84.3	77.6	81.9	91.9	82.7	86.0
IV	3.3	6.7	4.5	5.4	4.7	5.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	363	210	573	74	127	201
			Group IV			
I	—	0.6	0.3	—	—	—
II	15.2	17.8	16.5	17.9	20.0	18.7
III	83.2	78.9	81.0	82.1	80.0	81.3
IV	1.6	2.7	2.2	—	—	—
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	316	331	647	28	20	48

[1] In regard to social class I—IV cf. page 152.

longing to the social class III according to the official statistics, and the corresponding figure for fathers of children from all the groups. As to Groups I and II — that is, children placed under the care of the Board entirely or partly on account of

Table 32. **Distribution of the cases during the years 1926—1940 on social classes and the same distribution of the male population of Malmö, Hälsingborg, Landskrona and Lund according to official statistics of men, entitled to vote, for the years 1921, 1924, 1928, 1936 and 1940.**

Social class[1]	Fathers to children in group										Percentage number of men entitled to vote 1921—1940
	I		II		III		IV		Total		
	No.	%	No.	%	No.	%	No.	%	No.	%	
I	—	—	—	—	5	1.5	2	0.6	7	0.8	9.3
II	6	6.0	10	13.7	44	13.1	63	17.9	123	14.3	28.2
III–IV	94	94.0	63	86.3	288	85.4	286	81.5	731	84.9	62.5
III	66	66.0	58	79.5	268	79.5	277	78.9	669	77.7	.
IV	28	28.0	5	6.8	20	5.9	9	2.6	62	7.2	.
Total	100	100.0	73	100.0	337	100.0	351	100.0	861	100.0	100.0

[1] In regard to social class I—IV cf. page 152.

bad home conditions, in no instance did the father belong to social class I. On comparison with the normal figures, according to which 9 per cent of the population in the aforementioned towns belong to this social class, the difference is very great. This is probably because better-class parents have enough money to save their children from the exterior disadvantages of children from the poorer classes, and, consequently escape intervention by the Child-Welfare Board.

As will be noticed a certain percentage of the children in Group III (serious delinquents) as well as Group IV (criminal juveniles) came from the two higher social classes, but compared with the normal figures these two social classes are still underrepresented.

One would hardly expect any child whose father belonged to social class I or II would require taking in charge by the Board on account of the bad condition of the home, still, this has happened, although in relatively few cases, *i.e.*, 22 boys and 16 girls, 38 children altogether.

On further inquiries concerning these cases, however, it is found that some of the occupations recorded — *e.g.*, »agent»,

»ex-sergeant» and »inspector» — are rather difficult to classify, and belong to social class II hypothetically rather than in reality. In the single case from the first period, where the father belonged to social class I, and the home had been a contributory cause to intervention by the Board on account of slight delinquency in the boy, the family was one in which the father had died and the mother had gradually sunk financially as well as socially. Here too then, the family was placed too high for its condition at the time of intervention by the Board. It is most likely that this boy would never have required intervention at all if the father had lived.

It is quite evident, however, that many parents of the present children had a lower status than social class III; this is especially so during the later period for children in Group I, »risk of delinquency», where more than one-fourth of the children had fathers who were considered to belong to the lowest social class. In the earlier period the corresponding figures are not quite so high. This shift in the course of time may well indicate that the Child-Welfare Board has become more effective. The persons and children in question are largely roving subjects who were previously left to their fate.

On comparing boys and girls in respect of the social position of the fathers, we find — as so often before — that the girls are less fortunate than the boys. Here, however, the difference is relatively slight. It is most conspicuous in Group III where rather more of the girls come from the lowest social classes, *i.e.*, 86 % as against 82 % of the boys. But this difference is not very great, and, in view of the relatively small size of the material, it may be due to random variation. In Group III, however, more boys than girls come from social classes II and III, but this difference is not significant either.

On comparing the delinquent children (Group III) and the criminal juveniles (Group IV) in respect of the class from which they spring, we notice that the latter are to some extent from homes of a higher social standard. In both groups, it is true, about the same number of children come from social class III, but social class IV has furnished only half as many criminal

juveniles as delinquent children, in both periods alike, whereas social classes I and II have furnished more criminal juveniles than delinquent children.

GENERAL CHARACTER OF THE HOME.

In most cases, as pointed out before, the environment of the child in its first years of life is the parental home or the home of the mother. Apart from the external setting already touched upon — the social standing of the parents — it is obvious that a home may be bad or more or less good from many points of view, above all, the financial, moral and hygienic aspects.

In order to get an idea about the various standards of the homes, it is necessary to go through the various verdicts passed by the respective investigators. When there are several statements the author has summed them up in one general opinion. Some particular features will be discussed more fully later on.

It will be appropriate first to give a brief survey of the various categories under which the homes have thus been roughly classified. Home of type 1 covers such as have been characterized as »good homes», »orderly homes», »good and honest parents» or »nothing to be criticized in the home». Home of type 2 comprises the homes to which only slight objections could be made, which in most of the cases consisted in insufficient or no attention to the child; this often means homes where the mothers have to work for their living outside the home. Expressions such as »the child is left to fend for itself» has, when this is the only statement, relegated the home to this category, even though the expression possibly implies a more severe criticism. Finally, home of type 3 comprises the homes with all sorts of perceptible shortcomings. Naturally, homes characterized by terms such as »bad», »miserable conditions», etc. are entered under this category. This also applies to homes where one of the parents has been suffering from a protracted serious illness, and the home has thus sunk to a low standard financially as well as with regard to the attention paid to the children. The mere fact that the child has been shifted around

between various homes — *i.e.,* exposed to repeated changes in milieu — should not necessarily imply that the home is classified as »bad». Yet, several cases of this kind must come under this head, since this circumstance has led to intervention by the Board. There are also homes countenancing mental cruelty: *e.g.,* when a child born before the parents married and first reared by the grandparents and then taken over by its own parents, is treated like a »stepchild», or when its own mother »cannot bear the sight of the brat», or when the stepfather or stepmother uses all means to show the stepchild that it is an undesirable member of the family. But if one of the parents is entered in the penal register, this in itself has not detracted from the standing of the home if no other criticism has been made of the home. Even when the parents have committed some offence — before or after the intervention by the board — this need not necessarily lower the general standard of the home.

In the case of pronounced abuse of alcohol or immoral conduct on the part of the mother, however, it is hard to believe in any relatively high quality of the home, and such homes are therefore reckoned as grossly defective if the child is living with the parents. The same holds good if such misconduct occurs in the home on the part of the stepfather or stepmother and also foster-parents.

Naturally, homes about which no statement has been made — neither good nor bad — are reckoned as »unknown». It is quite possible that homes entered under category 2 — homes with minor objectionable features — have on account of the scanty data available been placed too high when they ought rather to have been entered under category 3 or registered as »unknown».

From a comparative survey of the total character of the homes in the various group of the material it is evident that children in Group I generally come from homes that have to be classified as category 3. This is only natural, as the very reason the children in this group have been taken under the care of the Board was the poor quality of the homes, which in practically all cases have been wanting in more than one respect.

It stands to reason, however, that when a child from such

a home is taken under the care of the Board, all the other children in the home become subject to the same measure, and hence several children in this group — boys as well as girls — are siblings. But, of course, the entire family of sibs is not always taken in hand, as some of the children may already have left home, or they may have passed the age limit or have been placed elsewhere and thus escaped intervention.

During the first period, 1903—1925, the 177 boys and 178 girls in Group I thus came from a total of 212 homes. A closer analysis of the features which in any one case have characterized these homes as unsuitable for the children to grow up in admittedly shows the criticisms to vary from one case to another, and yet certain abuses recur again and again.

The frequency of alcoholism in the home is particularly high in the first period, in which abuse of alcohol was recorded for 141 homes, that is, in two-thirds of the total. Both parents were addicted to drink in one-fifth of the cases (28), the mother alone in 36 cases, and the father alone in twice as many (76 cases). Besides this, drinking bouts occurred in several homes (13), even though abuse of alcohol was not recorded against any of these parents. For the same reason, the foster-parents were unsuitable in 5 cases.

The mother had been ill for a long time in 3 cases (tuberculosis). Mental affections were found in 7 mothers and 2 fathers, 2 mothers are characterized as »feeble-minded», and 8 mothers are taken to be »queer». In 62 cases the mother is stated to lead an immoral life, and in one-fourth of the cases the mother has taken to prostitution for a living.

Slovenliness, disorder and dirt are encountered widely, and over one-half of the homes are neglected. »No furniture, nor bedding», »rags and tatters everywhere», »children dirty and full of vermin», »running about half naked and ragged summer and winter». Vivid descriptions of this kind are found again and again.

To get an exact idea of the frequency of overcrowding in these homes is out of the question, of course, since direct statements are found only in the minority of the cases. Judging from the descriptions of the character of the dwellings they must have

been overcrowded as well as squalid throughout. »A miserable hovel», »an outhouse», »a back room without a kitchen or serviceable range», »a garret» are some of the remarks made by the inspectors illustrating the state of such homes.

Enmity between the parents, fights, quarrels and rows are recorded for 52 homes, and several of these homes have also been resorts for all kinds of rabble. Seven of the families are said to have been gipsies.

That the moral standard of these homes was also very defective is evident from the fact that a good many of the parents of these children have been in conflict with the law. As will be dealt with more thoroughly in the chapter on the characteristics of the parents, in the first period nearly 20 % of the fathers (24 out of 138 who could be found) and over 10 % of the mothers (21 out of 175) have committed crimes of a rather serious character.

In several cases the children have been taken in charge exclusively on account of apathy or ill-will on the part of the mother, even though conditions have not been so glaringly bad as in the above-mentioned cases. Mental maltreatment of the child has caused the Board to intervene in 6 cases.

In addition to all this more or less pronounced poverty probably prevailed in most of these homes, even though there are no direct or indirect statements to this effect. Besides 76 homes were incomplete on account of illegitimate birth; and half of them were broken up, while all of them suffered shortcomings of some of the kinds already mentioned. Only one father and two mothers were characterized as good.

In the following period, 1926—1940, the total number of homes for boys and girls of this group was 71. The external picture of the decay of these homes is perhaps not quite so striking, but the information about the parents is more exhaustive.

Terms such as »pimp», »boot-legger», »horse-dealer», »jail-bird» and »tramp» convey a more vivid impression of the »occupations» of the fathers than do the more civil designations of shoemaker, sailor etc. Over one-half of the fathers (34 out of 62 known cases) have served a sentence and the same applies

to one-fourth of the mothers (14 out of 59 known cases). Abuse of alcohol in the home is not quite as frequent here as in the preceding period; still, in every other home one or both parents are inebriates. The fact that the Temperance Board had to interfere in a majority of the given cases testifies to the correctness of the accusation. Nearly one-half of the mothers are considered to live an immoral life. Sexual intercourse between the mother and strangers in the presence of the child is reported not infrequently. Incest has occurred in 3 families, quarrels between the parents are part of the daily programme in many homes. Of the mothers, 2 are decidedly insane and 5 are characterized as »hypernervous» or »queer», while 3 of the fathers are markedly querulous.

A study of the various features presented by these homes in both periods indisputably attests that the character of these homes fully justify the designation »social bog», and that the legal »risk of delinquency» is a very mild expression for an environment as depicted here.

In the survey of the homes in Group II it has not been considered appropriate to put the boys and the girls under a common heading, as in most of these cases the behaviour of the child itself was the direct cause of intervention by the Board. It might be interesting to try to see whether there is any demonstrable difference between the homes of boys and girls in cases of minor delinquency also. In this group, too, some of the children are sibs. After exclusion of such cases we are left with 100 homes for the boys of this group in this period.

Nearly all these homes are also very poor, but this is not so pronounced as in the preceding group. The incidence of neglected homes is not so high here as in the preceding group, but it still amounts to one-fifth of the total. Abuse of alcohol occurs in every third home. The label »unsuitable home» is put on 10 homes without any particulars being given. Enmity and quarrels between the parents are recorded for at least every fifth home. Nearly one-fourth of the fathers (18 out of 75) have been sentenced for a relatively serious offence, and so have 6 out of 93 mothers, so that the quality of the mothers in this group seems somewhat better than in the preceding. In 10 cases

the mother is said to have turned to prostitution for a living. One-third of the homes are broken up, and just as many of the mothers are unmarried, so that the mothers of both categories have had to earn their living outside their home and thus have been unable to look after their children adequately. Several of these children have nowhere to go, but have to wait in the street or alley in all weathers till the father or mother comes home from work. The dwelling is cold, there is no fire in the stove, and the child has to fend for itself or turn to charitable neighbours. The remarks entered in the records by the inspectors testify strikingly to this: *e.g.,* »the boy is left alone, sits mostly on the steps, living on coffee and coarse bread»; »Mother dead. Poor home. When the father leaves for work in the morning, he cuts off a slice of bread for the children; in the winter no fire in the stove, and the children have to wait in the yard till the father returns».

In the following period, 1926—1940, the conditions in the home (60 cases) for the boys are about the same as above. Abuse of alcohol is not quite as frequent as before, but the percentage of criminal parents is higher, being 35 % for the fathers (19 out of 55) and 8.5 % for the mothers (5 out of 59).

Summing up, the homes of the boys may perhaps be said not to lie at such a low level in Group II as in Group I, and in many cases the shortcomings here consist in neglect of the children, carelessness and failure to shoulder parental responsibility.

As regard the girls in Group II, there is no demonstrable difference in the standard of the homes during the two periods, on which account it seems reasonable to treat them as one — in view of the small number. After ruling out a few sibs (3 cases) there remain 72 homes in all for both periods.

Abuse of alcohol is recorded for nearly one-half of the homes (33 out of 72), and three-fourths of the homes are characterized as »poor» or »very poor», several receiving municipal aid. One-half of the homes are »broken up», and one-fourth of the girls (19) were born out of wedlock. In 10 cases the mother has turned to prostitution for a living and supports in this way not only herself and her child, but, in two cases, also her

parents, who were aware of this and encouraged the trade. Squalor and discomfort are recorded for several homes, also quarrels and disagreements between the parents. Over 30 % of the fathers (19 out of 58) have served a sentence (14 fathers are unknown), and over 9 % of the mothers (5 out of 55) have been sentenced. In two families both parents have been sentenced.

On the whole it may be said that the homes of the girls in this group are alarmingly near the low standard of the home in Group I, and are decidedly worse than the homes of the boys in the same group.

In the remaining 2 groups (III and IV) of the material — delinquent children and criminal juveniles — a classification into various categories has been practicable in about two-thirds of the cases. Unfortunately there is no information about several of the homes from which these children came, especially in the first period, and in many cases the data are considerably more meagre than for the homes of the children belonging to Groups I and II. So in several cases the classification will be somewhat uncertain. (Cf. Table 33.)

The criticisms relegating the homes to category 3 have been of the same character as those in Groups I and II: neglect of the home, intemperance, immoral conduct and, last but not least, failure to care for the child. Several homes are directly designated as »bad» or even »wretched». Several families have had no permanent residence, moving about in the town as well as in the country. Two families are said to have been gypsies. Analysis of the crimesheet of the parents shows the frequency of sentenced parents to increase with the degree of badness of the homes. A feature common to several girls is that they have been shifted about between several homes — of step-parents, grand-parents and the respective mothers — or are living in homes with stepfathers.

Reckoned on homes from which data are available, in the first period nearly one-half (47.6 ± 3.1 %) of the delinquent boys (Group III) come from grossly defective homes, in the later period nearly two-thirds (57.7 ± 3.5 %) of the delinquent boys. In the first periods less than one-fifth (18.7 ± 2.4 %) come

Table 33. **Percentage distribution of the cases in groups III and IV according to the character of the home. Type 1 = good home, type 2 = medium home, type 3 = bad home.**

$P \pm \varepsilon(P)$ = percentage ± standard error of the percentage.

Type of home	Boys				Girls	
	1903—1925		1926—1940		1903—1940	
	No.	$P \pm \varepsilon(P)$	No.	$P \pm \varepsilon(P)$	No.	$P \pm \varepsilon(P)$
Group III						
1	50	18.7 ± 2.4	30	15.5 ± 2.6	24	13.9 ± 5.6
2	90	33.7 ± 2.9	52	26.8 ± 3.2	38	22.0 ± 3.1
3	127	47.6 ± 3.1	112	57.7 ± 3.5	111	64.1 ± 3.6
1—3.....	267	100.0	194	100.0	173	100.0
Unknown ...	129	32.6 ± 2.4	33	14.5 ± 2.3	74	30.0 ± 2.0
Total	396	—	227	—	247	—
Group IV						
1	103	36.8 ± 2.9	103	41.0 ± 3.1	8	19.0 ± 6.1
2	67	23.9 ± 2.5	55	21.9 ± 2.6	15	35.7 ± 7.4
3	110	39.3 ± 2.9	93	37.1 ± 3.0	19	45.3 ± 7.7
1—3.....	280	100.0	251	100.0	42	100.0
Unknown ...	44	13.6 ± 1.9	117	31.8 ± 2.4	11	20.8 ± 5.6
Total	324	—	368	—	53	—

from good homes, but in the last period the number of such boys was lower (15.5 ± 2.6 %).

In both periods the percentages of criminal juveniles coming from good and bad homes are about the same. A comparison of the homes of the delinquent boys thus shows that a greater portion of the criminal boys have good homes. The difference in this respect for the first and later periods amounts respectively to 8.3 ± 4.2 % and 20.6 ± 4.6 %, and is the later statistically significant. This difference seems to indicate that delinquent children more often come from really bad homes than do criminal juveniles. In my opinion this must be attributed to the way in which society reacts towards delinquency and crime respectively. The individual guilty of a criminal act is sentenced, regardless of his home condition, whereas a really delinquent child is not always turned over to protective education. If the

latter has a good home, and his parents are considered capable of correcting him themselves, the Board may often dismiss him with relatively mild measures as for instance a caution.

In both periods the girls of Group III came from bad homes in about two-thirds of the cases. In the first period only a few girls (10.5 %) came from really good homes, but in the later period a somewhat higher percentage (15.5 %) had had the benefit of a good home. But this difference is not very great and not statistically significant.

On comparing boys and girls in this category, is found that, in the first period, the markedly bad homes were more common among the girls, and likewise that the boys more often came from really good homes. In the later period the difference is less pronounced. A few more girls have really bad homes, but, on the other hand, a few more girls now come from good homes (one-third of these homes were not parental homes, however, but foster-homes). But the criminal boys come considerably less often than the girls from good homes.

FINANCIAL FACTORS.

One of the crucial factors in the character and standard of the home is the financial status. So it may prove of interest to analyze the homes more thoroughly from this point of view, and try to get a reliable idea as to whether these homes are worse off financially than the homes of the average population. Naturally, no exact data are available as to the family breadwinner's income, how many he is obliged to support etc. All we have to go on are the direct statements by the inspectors as to the existence of poverty, and the official registers as to whether the respective families have been in receipt of poor relief. If, to maintain an objective standard, we take into account only the latter data, it is obvious that the findings will be minimum figures.

For the sake of completeness, however, an attempt has been made to estimate the degree of poverty of the various homes in this material, according to their description by the

inspectors. This estimation is based on expressions such as »the children starve and are in a bad way» or »the children are ragged and dirty». I have also made use of such statements as »the dwelling consisted of a »rickety shack» or »a partition in a municipal barrack» and other descriptions indicating that the home cannot have been other than poor.

From Table 34 it is evident that many of the children in Groups I and II come from homes that were not only bad but also poor. This holds good for the boys as well as for the girls, and it applies to both periods.

As regards the boys in Groups III and IV — delinquent and criminal boys respectively — their homes in the first period are about equal in this respect, and one-third may be designated as poor. In the later period the homes of the criminal boys are on an average more well-to-do, and only about one-sixth of these boys come from poor homes. For the girls in Groups III and IV these conditions are largely the same in the first period, whereas in the period of 1926—1940 the poor homes are more common among the girls.

On comparison of the homes of the boys and the girls — in all the groups together — poor homes are found to be more common among the girls than among the boys. The difference for the two periods is 20.6 ± 3.0 % and 21.6 ± 3.6 % respectively, *i.e.* significant. Turning to the frequency of paid out relief, we find a distinct difference between the earlier and the later periods. In the first period such relief may be said to have been distributed almost equally between the various groups in the material, and hardly more to the markedly bad homes than to the others, whereas in the later period the aid been given considerably more often to the latter. It seems reasonable to expect that in the earlier period the granting of relief was dictated by moral points of view. In the case of Group III, children with pronounced tendency to delinquency, relief has likewise been granted somewhat more often in the later period than in the first — which points in the same direction.

In the first period the percentage of homes that were given such aid was lower than the average percentage for relief to families in Malmö, which in 1903—1923 amounted to 5.1 %.

Table 34. **Frequency of children from poor homes.**

Group	Total No. of children = N	Children from homes recorded as poor by the Child-Welfare Board		Of these, children from homes having got poor relief		Children from homes being poor according to additional information		Total No. of children from poor homes	
		No.	% of N	No.	% of N	No.	% of N	No.	% of N
				Boys 1903—1925					
I	177	34	19.2	7	4.0	114	74.6	148	83.6
II	105	31	29.5	9	8.6	42	40.0	73	69.5
III	396	90	22.7	13	3.3	40	10.1	130	32.8
IV	324	102	31.5	12	3.7	13	4.0	115	35.5
Total	1002	257	25.6	41	4.1	209	20.9	466	46.5
				Boys 1926—1940					
I	69	25	36.2	15	21.7	33	47.8	58	84.1
II	61	22	36.1	14	23.0	15	24.6	37	60.7
III	227	46	20.3	17	7.5	17	7.5	63	27.8
IV	368	54	14.7	12	3.3	5	1.4	59	16.0
Total	725	147	20.3	58	8.0	70	9.7	217	29.9
				Girls 1903—1925					
I	178	27	15.2	8	4.5	127	71.3	154	86.5
II	52	14	26.9	2	3.8	24	46.2	38	73.1
III	87	18	20.7	3	3.4	12	13.8	30	34.5
IV	30	8	26.7	3	10.0	3	10.0	11	36.7
Total	347	67	19.3	16	4.6	166	47.8	233	67.1
				Girls 1926—1940					
I	66	34	51.5	21	31.8	26	39.4	60	90.9
II	23	10	43.5	3	13.0	6	26.1	16	69.6
III	160	32	20.0	11	6.9	26	16.3	58	36.3
IV	23	4	17.4	2	8.7	2	8.7	6	26.1
Total	272	80	29.4	37	13.6	60	22.1	140	51.5

For the period from 1928—1938 this percentage amounted to 5.8 %, and the percentage for families in the present material who received such aid was higher than the average percentage, and was considerably higher for Groups I and II.

Unfortunately fully comparable figures are not available from the official statistics, but with the above-mentioned figures as basis it seems as if homes receiving poor relief are over-represented in this material.

The closest companions of poverty are reckoned to be scanty house-room and the lodging system. Direct information about the frequency of these two conditions is scarce in the present material.

In both Groups I and II, in which bad homes are most pronounced, overcrowding is recorded for 10 % of the homes in both periods, whereas the frequency of this condition is not quite 5 % for the direct cases of delinquency in the first period, and somewhat higher in the later period. About the same applies to the homes of the criminal juveniles. Overcrowding appears to have been more frequent in the homes of delinquent girls than in the homes of delinquent boys, here reaching nearly 10 %. Naturally no data are available on the number of rooms in the home and the members of the family, so that an exact calculation is out of the question. For comparison purposes, it may be mentioned that, at the special investigation concerning the frequency of scanty house-room that was carried out in 1933, it was found that practically one-half of all the dwellings in the towns covered by this investigation were overcrowded. So the figures obtained in this material seem incredibly low and must be based on another standard for the estimate. The data on the number of lodgers are too incomplete to allow of any statistics.

Another common companion of poverty is squalor, slovenliness and disorder, *i.e.*, neglected and mismanaged homes. Such a condition was found in over 60 % of the homes belonging to Group I in the first period; and even if conditions are not quite as bad in the later period, still one half of the homes under this category are disorderly and neglected. In this respect, even the homes from which the delinquents proper came are bad, but to nothing like the same extent as the aforementioned. It is recorded that dirtiness and disorder were found in 17 % of the homes of these children (III), equally often in both periods and about just as often for boys as for girls. In this respect,

too, the homes of the criminal juveniles (Group IV) were better than the others, with only about 5 % neglected homes in the later period, but rather more in the first period.

It seems fairly probable that poverty may lead to dissension and quarrel betwen the various members of the family. Data on the occurrence of enmity between the parents, quarrel and fighting are relatively frequent, especially in the group of bad homes (Group I), where they amount to as much as one-fourth of the total. Among the cases of real delinquency this tragic state of affairs is considerably less frequent, even though it is reported in 7—8 % of the cases, and for the girls in one of every ten homes.

On the whole it may be said that this investigation into the environment in which the children have grown up shows that a great majority of the homes here concerned present alarming defects such as poverty, poor hygienic conditions and other causes of domestic discomfort and misery.

SIBLINGS.

If the picture of the environment of the children is to be complete we cannot ignore the other children who have grown up in the same home. Our investigation into this aspect of the matter falls short on several points, however. For one thing, it has not always been possible to obtain information about the number of sibs either at the time of intervention by the Board or at all, because data on this point are very often missing in the records, especially from the first period. Nor has it always been possible to find the given addresses in the parish registers. Thus, it has not been possible to obtain any information of this kind for 5 % of the children. Furthermore, sibs have not been looked up systematically in the register of the Child-Welfare Board — and even less in the penal register. Thus, the data here presented are derived exclusively from the statements in the records for every child. Still, it may be of some interest to get an approximate idea of the extent to which the children in this material come from families with many children.

In this compilation of the sibling groups, the children about whom no data on this point have been obtained are left out of consideration. Further, the only child of an unmarried mother is also ruled out. On the other hand, if an illegitimate child is living with its mother and stepfather, it is included, as also are its half-brothers and half-sisters, if any. Of course the same applies to children from broken homes, living with one of the parents.

It can be established at once that the delinquent child does not to any great extent represent the only child of a family. The percentage of only children varies between 3 and 5 %. An exception to this rule is found in the boys of Groups I and II, who, strange to say, were without brothers or sisters in 13 % (both groups). The »only child» is generally taken to be spoilt, the object of excessive attention on the part of the parents, but this does not seem to apply to the present material. On the contrary, it looks as if the only child has rather been regarded as most unwelcome, or often been maltreated mentally. This material accordingly gives no support whatever to the view advocated by BURT, that the delinquent child »again and again is found to be an only child».

It is more difficult to say anything about the other extreme — that delinquent and criminal children largely belong to families with many children. It depends, of course on the number of children implied by »many». What was looked upon in the first period as a relatively small family — 3 or 4 children — is now taken as a large one. For the early period it has not been possible to obtain any comparative figure for the average number of children in the families of the rest of the population. A rough estimate of the matter in this material shows that in the first period one-fourth of the delinquent boys had at least 6 sibs. The criminal boys in particular had many sibs, as two-fifth of them were recorded to have at least 6 sibs.

In the later period the number of sibs has decreased, so that less than 15 % of the present children come from families with at least 7 children. The criminal boys are still leading in this respect, as more than 20 % of them have at least 6 sibs.

If, further, we compare the average number of children per

family for the delinquent as well as for the criminal ones with the average number of children per family in Malmö in 1930, namely, 3.3 — which already in 1936 has fallen to 2.5 per family — it may still be reasonable to claim that the boys in the present material belong to families with relativerly many children, when on an average there were between 3.1 and 3.5 children in their respective families.

A survey of the sibs of the delinquent as well as of the criminal boys yields quite a high incidence of asocial or criminal acts. In the first period nearly one of every four boys in Group III (23.5 %) had one or more sibs who had been in contact with the Child-Welfare Board or the Court of Justice. In the following period this frequency has decreased somewhat, as now only one of every five boys has had one or more sibs who were entered in the records of the Child-Welfare Board or the Court. Nearly the same figures apply to the court clientele (Group IV). Still, the later period shows a relative decrease in that only 11.1 % of the boys have sibs who have been sentenced or taken in charge by the Board.

The delinquent girls (Group III) have had relatively fewer criminal or asocial sibs, the figures being only 15 % for the first period, and 12 % for the later. But criminal sibs are more frequent for the criminal girls (Group IV), however, as 26 % of these girls had brothers or sisters who had gone the same ways as they had themselves. Still, the number of criminal girls is too small to allow of any estimate.

At a first glance these figures may be thought to suggest that hereditary factors are of no significance in this connection. It is obvious, however, that if the environmental factors cause children to become delinquent and criminal, they ought to mean the same to the sibs and cause many of them to go the same way as the subjects under examination in this material. The figures presented here, however, are to be accepted only with reservations. For, as mentioned already, no systematic investigation has been made into the fate of the sibs.

Summing up, then, we can say that the children in this material less often had the chance of growing up in the care of both parents than children in general, and that the girls were even

more unfortunate in this respect than the boys: this is particularly marked in group III — children showing more serious delinquency. A number of the homes whence these children come are incomplete, either due to illegitimate birth or because they have been broken up; in both these respects the girls are worse off than the boys. The children have often had to change their home, the girls more frequently than the boys. More girls than boys also seem to come from the lowest classes of the population. The total character of the homes is in most cases extremely bad, and if any difference of degree can be made, the homes of the girls are worse than those of the boys. Of the boys in group III (more serious delinquency), $^1/_3$ come from good homes, while this can only be said of far fewer girls. In a number of cases the homes could be designated 'poor' or 'very poor': this, too, is true of more girls than boys. The girls are thus considerably handicapped in a number of respects; this applies both to the material as a whole and, in particular, to group III (more serious cases of delinquency). In other words, the environmental influences of the girls during childhood are consistently worse than those of the boys.

Before discussing more fully the conceivable cause of this difference, we must touch briefly on the extent to which the delinquency of the girls is of an other character than that of the boys. Girls, of course, are not so prone as boys to violent actions and more serious forms of robbery, e.g. burglary. They are handicapped by inferior physique. The crimes against property that girls commit usually take the form of pilfering, where girls have as great possibilities as boys, if not greater. The bulk of the girls' delinquency takes the form of sexual offences, however.

It should, however, be noted that girls falling into bad ways are less well equipped intellectually than girls in general, and are worse off in this respect than delinquent boys. This is shown in a later chapter. That is to say, the delinquent girls are less well provided than the delinquent boys with that protection against asocial conduct which a normal intellect is thought to constitute. It might most reasonably be expected, then, that even a slight deterioration of environment would be

enough to drive the girls into bad ways. The exact opposite is the case.

Without touching closely on that interplay between genes and environment which is the necessary prerequisite of all human action and behaviour generally, we may yet quote the following as debatable causes of the observed difference in environmental influence between delinquent girls and delinquent boys: It is conceivable that the girls are less hereditarily prone to delinquency than the boys, and therefore require greater influence from their environment to fall into bad ways. In other words certain genes, whose presence in an individual results on contact with certain environmental stimuli in qualities leading to crime and delinquency, might be sex-linked. Other characters such as prudence, timidity, lack of enterprise etc., which have always been considered particularly feminine — no matter whether they are conditioned by hereditary or the result of early influence of environment — might be thought to be a guard both against delinquency and criminality, and girls only fall into bad ways when both these qualities and the intellectual equipment fail, and the external surroundings are particularly unsatisfactory. It thus seems as though boys are more sensitive to the influence of environment, and fall into bad ways even when this is only relatively bad, whereas far greater and harder pressure is needed in the case of girls.

CHAPTER VII.

CHARACTERS OF THE PARENTS.

The description of the homes of the children gives a good deal of indirect information about the characters of the parents. It is the parents, indeed, who put their stamp on the home and determine the milieu in which the children grow up. Naturally, a more thorough study of the parents, not only as educators, but also from other points of view, would have been desirable. But there is not much direct data on the tendencies and characters of the parents in the records: they are either vague and uncertain, or more or less subjective.

However, an attempt has been made to ascertain certain features of the parents by searching for information of a more objective kind, i.e. the addiction of the parents to criminality and abuse of alcohol. An investigation into these aspects has the advantage that a comparative material is available as far as the average population is concerned in the investigations reported by DAHLBERG (1942, 1943). Finally it has been computed how often the records say that the parents have been good and honest.

Tables 35 and 36 show to what extent the parents had up to January 1st 1944 been guilty of some criminal offence for which they had been brought before a court and sentenced. This tabulation is based on data obtained from the Central penal register, therefore comprising only more serious cases, i.e., crimes involving imprisonment, besides sentences for theft and petty larceny, even though the punishment has been milder. From the investigation submitted by DAHLBERG (1943) it is evident that the total risk of a sentence of this kind at the age of 70 years for men in Malmö amounts to 10.2 per cent, as calculated by

Table 35. **Number of cases with criminal fathers.**

Sex and period	Group	Total number of cases	No. of fathers not looked for in the Register of criminals because		Number of fathers looked for in the Register of criminals	of these, criminals	
			not identified	»unknown»		No.	%
Boys 1903—25	I	177	2	37	138	30	21.7
	II	105	1	24	80	20	25.0
	III	396	12	55	329	41	12.5
	IV	324	26	25	273	28	10.3
Boys 1926—40	I	69	—	4	65	38	58.5
	II	61	1	4	56	20	35.7
	III	227	—	14	213	54	25.4
	IV	368	21	26	321	67	20.9
Boys 1903—40	I	246	2	41	203	68	33.5
	II	166	2	28	136	40	29.4
	III	623	12	69	542	95	17.5
	IV	692	47	51	594	95	16.0
Girls 1903—25	I	178	13	46	119	18	15.1
	II	52	—	10	42	14	33.3
	III	87	6	23	58	7	12.1
	IV	30	1	3	26	4	15.4
Girls 1926—40	I	66	—	5	61	31	50.8
	II	23	—	4	19	6	31.6
	III	160	4	25	131	36	27.5
	IV	23	1	2	20	3	15.0
Girls 1903—40	I	244	13	51	180	49	27.2
	II	75	—	14	61	20	32.8
	III	247	10	48	189	43	22.8
	IV	53	2	5	46	7	15.2

the conditions in 1937. At the age of 50 the corresponding figure is 9.3 per cent. So the risk increases only slightly after the age of 50. As pointed out by DAHLBERG, it is rare for a person to commit his first crime after this age — but this does not imply, of course, that men in their fifties are rarely criminals. This risk percentage may now be applied for comparison with the figures obtained in our material. It should be pointed out that in many of our cases the parents have died before the age

Table 36. **Number of cases with criminal mothers.**

Sex and period	Group	Total number of cases	No. of mothers not looked for in the Register of criminals because		No. of mothers looked for in the Register of criminals	of these criminals	
			not identified	»unknown»		No.	%
Boys 1903—25	I	177	7	1	169	19	11.2
	II	105	7	—	98	7	7.1
	III	396	23	1	372	14	3.8
	IV	324	44	2	278	12	4.3
Boys 1926—40	I	69	2	—	67	17	25.4
	II	61	2	—	59	5	8.5
	III	227	13	1	213	10	4.7
	IV	368	38	3	327	13	4.0
Boys 1903—40	I	246	9	1	236	36	15.3
	II	166	9	—	157	12	7.6
	III	623	36	2	585	24	4.1
	IV	692	82	5	605	25	4.1
Girls 1903—25	I	178	49	2	127	22	17.3
	II	52	17	1	34	3	8.8
	III	87	22	2	63	4	6.3
	IV	30	3	—	27	2	7.4
Girls 1925—40	I	66	15	—	51	10	19.6
	II	23	3	—	20	3	15.0
	III	160	21	—	139	11	7.9
	IV	23	2	—	21	1	4.8
Girls 1903—40	I	244	64	2	178	32	18.0
	II	75	20	1	54	6	11.1
	III	247	43	2	202	15	7.4
	IV	53	5	—	48	3	6.3

of 50, or belong to a younger age class. This applies in particular to the later part of the material.

Such information about the fathers was obtained in 1951 cases — or 96 % of the known fathers. Naturally, several fathers have been »unknown», i.e., the paternity has not been established (307 cases) and in a few cases (13 in all) the children have been »offspring of unknown parents», i.e., both parents have been unknown.

Of the fathers who could be investigated in this way, 417 were found in the penal register (21.4 per cent). This figure is more than twice as high as the above figure given by DAHLBERG (9.3 %) for the men in Malmö in 1937. For the above-mentioned reason the difference between DAHLBERG's and my figures should actually be somewhat higher.

Considering the fathers of the boys in the various groups it is plainly evident that Group I, the group of decidedly bad homes, are most heavily encumbered in this respect, espicially in the later period when more than one-half of these boys had fathers who had been sentenced for some crime. In the first period, on the other hand, we find only one sentenced father to every fifth boys.

In Group II, in which the homes constitute a contributary factor in the intervention of the board. there is one sentenced father per 4 boys in the first period, in the later period per 3 boys.

In Group III, serious cases of delinquency, the number of sentenced fathers is smaller than in the preceding groups. Still, in the later periods there was a criminal father per 4 boys.

The criminal juveniles (Group IV) have better fathers than have the delinquents — as shown before in some other connection — since in the later period only one out of every fifth boys has a criminal father, and in the first period the incidence of a criminal father is even lower.

The material for girls is considerably less than that for boys, so that it is hardly worth dividing it into groups. If girls and boys are compared with the help of weighed figures for the girls, so that they become comparable with the figures for the boys, the difference is found to be slight. The mean for the boys is 20.2 per cent, while for the girls the mean is 21.3 per cent. In view of the standard error, the difference 1.1 ± 2.1 % must be attributed to chance. (Cf. Table 35.)

It will be seen, then, that the frequency of punished fathers is highest in the two groups in which the homes on an average are worst, and lowest in Group IV which has the highest number of good homes. An exception to this rule is admittedly found in Group I, for the boys as well as the girls, in the first

period, in which the ratio of punished fathers is somewhat lower, but presumably this is a random variation. In view of the preceding findings concerning the differences of the character of the homes, it seems justifiable to say that the worse the homes, the higher the ratio of criminal fathers — which really is not to be wondered at.

Further, on comparison of the two periods, 1903—1925 and 1926—1940, the frequency of criminal fathers is found to be higher in the later period. This applies to the boys as well as to the girls. For the boys this ratio in the two periods is 14.5 % and 27.3 % respectively, for the girls the weighed figures are 15.9 % and 24.0 %. One would rather have expected the converse, as the fathers are older in the first period than in the later, in which about one-third were not 50 years at the time of the investigation. It is probable that these figures are to be interpreted as evidence of a rise in the criminality during the later years.

Of the mothers of these children, 2065 were investigated, that is 88.5 % of the total material, as the afore-mentioned 13 children both of whose parents were unknown, are left out of account here (cf. Table 36).

The total number of punished mothers is 153, or 7.4 %, which is five times higher than the total risk for women of 50 years that is found by DAHLBERG (1943) to be 1.5 %. The latter figure applies to the population of Stockholm, but in view of the figures obtained for the men it seems reasonable to assume that, as far as criminality is concerned, Stockholm and Malmö keep at the same level.

On investigation of the mothers of the boys, we find — just as for the fathers — that the ratio of criminals is highest in Groups I and II in both periods. On the other hand, there is no difference in this respect between the mothers in Groups III and IV, criminal mothers being about equally frequent in the two groups and, in comparison with the normal figures, only a little more than twice as frequent as in the normal population. In contrast to this, the frequency of criminal mothers among the boys in Group I as compared to the normal figures is alarmingly high, for in the later period one-fourth of the

mothers have been punished, as against 1.5 per 100 women within the average population.

As regards criminality, the mothers of the girls seem to show higher figures than those of the boys. The figure for the boys is 6.1 per cent, while the girls yield a weighed mean of 9 per cent. The difference is 2.9 ± 1.4, and is thus not significant. A larger material is required to illustrate this matter. So, on the whole, it is evident that as regards to criminal parents, these children are worse off than other children, maternally as well as paternally. With reference to the low percentage of punished women in the average population, which according to DAHLBERG is only one-tenth of the male percentage, it is also safe to claim that the mothers of the children in the present material are worse equipped. In the compilation above, the parents may figure twice or more, as the children are reckoned as statistical units. If the fertility of criminal parents is the same as that of the general population, of course, it makes no difference how we compile the figures. As no definite data are available concerning this point, however, it seems reasonable to assume that the difference cannot be very great, if any.

It is interesting to compare the above findings with the data as to the state of the homes. The question arises: how many delinquent children from good homes have criminal parents? From Table 33 it will be noted that, considering both periods as one, the boys in Group III came from good homes in 80 cases. Of the respective fathers 8 had been punished for some crime (out of 70 fathers investigated), but no mothers (70 investigated). Of the criminal boys, 206 came from good homes. Of the respective parents, 19 fathers had been punished (179 investigated), and 3 of the mothers (177 investigated). Considering these two groups as one, we then have 286 boys from good homes. In 27 of these cases, however, the fathers had been punished, but in the great majority of the cases there was nothing of the kind on record for the fathers. In the 3 cases in which the mothers had been criminals nothing was entered against the fathers, so that none of these homes was encumbered in both ways.

Among the girls in Group III, in both periods, only 24 came

from good homes — and 3 of these were not their own homes but good adoptive or foster homes. Neither parent of the girls coming from their own homes had been punished (all investigated). For the girls from foster or adoptive homes, none of the investigated mothers (8) had been punished, while one father (out of 6 investigated) had a sentence.

Among the criminal girls (group IV) 8 came from good homes, and none of these parents had been punished.

ABUSE OF ALCOHOL AMONG THE PARENTS.

Information as to the addiction of the parents to drink has been obtained in part from the records, in part from the Temperance Board in Malmö. The former information suffers from not being comparable with the data for the general population; besides which it is not uniform, as the individual cases may have been estimated differently. On the other hand, the information obtained from the Temperance Board may be looked upon as uniform and above all, comparable with the data for the total population. But the Temperance Board did not start functioning till 1917, and during the first years its activity may be said to be somewhat ineffective. For this reason, the data obtained from the records and the Temperance Board are tabulated separately.

It should be mentioned that only the fathers have been looked up in the register of the Temperance Board, not the mothers. Here, enquiries was made about the same number of fathers as in the penal register.

Comparing the information obtained from the Temperance Board with the data from the records, we find a considerable discrepancy in the first period, the data from the records give considerably higher figures than does the information from the Temperance Board, while in the later period the reverse is the case.

We may assume, that in the first period the data in the records come near the truth — especially as they have often been verified by enquiry among the neighbours. Consequently the

Table 37. **The children distributed with regard to their parents being inebriates.**

Group	Fathers					Mothers		
	Total number	Inebriates according to the Temperance Board		Inebriates according to the Child-Welfare Board		Total number	Inebriates according to the Child-Welfare Board	
		No.	Per cent	No.	Per cent		No.	Per cent
				Boys 1903—1925				
I	140	18	12.9	66	47.1	176	48	27.3
II	81	13	16.0	24	29.6	105	7	6.7
III	341	26	7.6	48	14.1	395	5	1.3
IV	299	14	4.7	54	18.1	323	5	1.5
				Boys 1926—1940				
I	65	29	44.6	7	10.8	69	4	5.8
II	57	19	33.3	8	14.0	61	2	3.3
III	213	39	18.3	19	8.9	226	1	0.4
IV	342	59	17.3	17	5.0	365	—	—
				Girls 1903—1925				
I	132	13	9.8	57	43.2	176	57	32.4
II	42	4	9.5	18	42.9	51	8	15.7
III	64	4	6.3	6	9.4	85	3	3.5
IV	27	5	18.5	—	—	30	—	—
				Girls 1926—1940				
I	61	34	55.7	4	6.6	66	6	9.1
II	19	7	36.8	2	10.5	23	1	4.3
III	135	19	14.1	5	3.7	160	1	0.6
IV	21	5	23.8	—	—	23	—	—

result ought to be that in this period nearly one-half of the boys in Group I had fathers addicted to drink. The same applies to one-third of the boys in Group II, and also to one-fifth of the boys among the criminal juveniles (Group IV). Of the delinquents (Group III) not quite so many had fathers that could be entered as alcoholists (cf. Table 37).

If in the later period we take into account only the data obtained from the Temperance Board, it is evident that the

highest frequency of alcoholists among the fathers is found in Groups I and II, comprising children from particularly bad homes. On comparison of the ratios for these two groups with the figures of risk as ascertained by DAHLBERG (1941) for the male population of Malmö — namely 10.8 % for the period of 1929—1933, and 14.1 % for the period 1934—1938 at the age of 70 and 8.0 % respectively 9.3 % at the age of 50 — it is plain that men addicted to drinking are 3 times as common among the fathers of these children than among men on an average in the town.

Group III and IV show no difference in the frequency of fathers regarded as alcoholists, this category being equally common in both groups. So in this respect the criminal juveniles cannot be said to have been better off than the delinquents — in contrast to so many other respects. Compared with Dahlberg's figures the difference from the general population is not so great here. The incidence of alcoholism among the fathers in these two groups is only half as much again as in the total population.

If the ratio of fathers addicted to drink is calculated for all groups together in the last period, according to the data received from the Temperance Board, it again looks as if the girls were worse off than the boys, having relatively more fathers addicted to drink. Among the fathers of the girls 24.8 % were alcoholists (weighed figures), as against 21.6 % for the boys. The difference amounts to 3.2 ± 3.2 % but is not statistically established.

Some of the mothers of these children have also been addicted to the abuse of alcohol. As mentioned already, the information about these mothers has been obtained exclusively from remarks in the records, and thus it cannot be said to be complete. With this reservation however, it can be seen from Table 37 that drinking mothers were distinctly more common in the first period, especially in Group I, for girls as well as for boys. It is further evident that »booze-fighters» are more common among the mothers of the girls.

Other characters, undesirable genetically as well as environmentally, are naturally to be found among the parents of these

Table 38. **Mental qualities in the parents.**

Quality	Boys				Girls			
	Group							
	I	II	III	IV	I	II	III	IV
Fathers								
Total number	205	138	554	641	193	61	199	48
»Brutality»	18.5	12.3	4.2	1.6	5.7	3.3	1.5	—
Debility	0.5	—	—	—	—	—	—	—
Psychopathy	2.0	2.2	—	0.3	1.0	—	0.5	—
Insanity	2.0	1.4	1.3	0.6	1.6	—	2.0	—
Having committed suicide	2.4	3.6	1.1	0.3	2.1	—	—	—
Mothers								
Total number	246	166	623	692	244	75	247	53
Prostitute	36.7	13.3	3.2	0.6	40.5	16.2	5.7	—
»Brutality»	6.5	1.8	1.1	—	3.3	2.7	—	—
Debility	6.9	1.2	0.5	0.6	5.0	—	1.6	1.9
Psychopathy	2.4	1.2	0.2	—	2.1	1.4	1.2	1.9
Insanity	2.4	2.4	1.9	0.7	3.3	—	0.8	—
Having committed suicide	—	—	0.2	—	0.8	—	—	—
Hysteria	1.2	3.0	2.1	1.5	0.8	1.4	2.4	—
Nervousness	1.2	1.8	3.2	0.6	4.1	4.1	2.9	—
Epilepsy	0.4	—	—	—	—	—	0.4	—

children. A character which, next to criminality and alcoholism has to be assigned considerable significance as a factor in delinquency, is the occurrence of bad sexual habits among the mothers. Quite naturally, information on this point may not be obtained from official sources, so that we are limited to any possible remarks put down in the records. It may be taken for granted, however, that when such data are encountered in official records, they may be looked upon as fairly reliable, especially as most of these data are obtained from the police, while in most other cases they have been verified through enquiry among the neighbours. The tabulation of these data shows once more a strong encumbrance of Group I in which over one-third of the mothers in both periods, for the boys as well as for the girls, were addicted to loose habits. (Cf. Table 38.) In this respect the mothers of the criminal juveniles were

somewhat better than those of the delinquents. The encumbrance was somewhat greater for the girls than for the boys but the difference is not statistically significant.

Characters such as debility and psychopathy have also been recorded, but the figures are too small to be assigned any importance. It should be mentioned, however, that 23 of the fathers committed suicide — that is, somewhat over 1 per cent of the total number of known fathers (2 039) — and that 24 fathers were insane.

Of 2 333 mothers 37 were recorded as insane (1.6 per cent), and 42 were put down as mentally retarded (1.8 per cent), while 24 (1.0 per cent) are stated to have been »psychopathic».

Even though evidently a majority of the parents of these children in one or more respects have shown unfavourable characters, we also meet with remarks to the effect that the parents are known to be good and honest. Naturally, however, such remarks are very scarce. For one thing it is always easier to obtain information about bad parents. Furthermore, several of the parents have died, and as a rule the neighbours knew nothing about them — otherwise the information was most often obtained from the neighbours. The children in Group I, boys and girls together, are worst off in this respect. Only 2 of the mothers in this group are put down as being »good», and in one case the father is said to be »orderly», but in no instance is anything good said about both the parents. In Group II, among the girls, the father is designated as an »honest working man» only in one case, while the boys in the same group presented »orderly» mothers in 4 cases and »industrious» fathers in 3 cases; but even among these children there is no case in which neither parent had been given a bad mark.

Groups III and IV naturally have the greatest number of good parents, and yet they are but relatively few. Thus, considering both periods as one, in Group III both parents of the boys were characterized as »honest and industrious» only in 25 cases (4 per cent), while 21 fathers (3.4 per cent) and 27 mothers (4.4 per cent) were looked upon as »diligent and efficient». Among the girls in the same group, both parents were

good in 12 cases (4.9 per cent), while the mothers were good in 6 cases (2.4 per cent), the fathers in 4 cases (1.6 per cent).

As in several other respects concerning the home conditions, the boys in Group IV (criminal juveniles) were best off in this respect too, as in 34 cases (4.9 per cent) both parents were recorded as »honest and industrious», while 29 mothers (4.2 per cent) and 49 fathers (7.1 per cent) were put down as »orderly».

The above-mentioned information is of varying value. Data on criminality and alcoholism may be taken as more reliable, especially because such estimates are made along fairly uniform lines when it comes to crimes and intervention by the Temperance Board, and they are comparable with such data for the population in general. Other data are of a more supplementary value, as we have no corresponding comparative material, and this kind of data is based in some degree on personal judgments.

The factual data show that, with reference to the characters of the parents, these children are far worse off than average children. This is of great practical interest. From a theoretical point of view, however, the meaning of these figures is rather obscure. This suggests that the home conditions have been particularly unfavourable to the children, and that the parents have been a bad example to them — something which may be of importance. Possibly, perhaps, the bad characters of the parents may signify a poor hereditary quality, so that the children inherit some rather unfavourable genes and thus readily go astray. Finally, it has to be pointed out that the poor qualities of the parents may also imply counter-suggestion. Children react to the poor behaviour of their parents. Thus, for instance, DAHLBERG (1946) has shown that a not inconsiderable number of teetotallers (15 per cent) have become so because their parents or other near relatives had been alcoholists or inclined to pronounced abuse of alcohol.

CHAPTER VIII.

DISTRIBUTION OF THE HOMES ON DIFFERENT REGIONS OF THE TOWN.

Slum districts such as are found in some foreign cities and have been long considered as hearth and hot beds of crime and delinquency, hardly exist in any Swedish town. Still, certain areas in some districts of the town, where buildings have become antiquated, and where sanitary fixtures now generally considered indispensable are still lacking, have declined to a rather low standard, and consequently the rents have become cheap thus attracting economically unfortunate persons. They have thus ofter become the meeting place for more or less dubious elements. Such areas are not established definitely, however, but may change in appearance as well as character through the erection of new buildings and exchange of residents. Nor is it always the majority of houses in one area that may be charcterized as entirely out of date and badly inhabited. In most instances this applies only to certain streets, or even only to certain houses scattered about. Consequently it would be difficult to point out any particular part of Malmö as a slum district — specially as such areas in the long period covered by the present investigation, 40 years, would have changed their character several times. For a long time, however, certain areas in the eastern part of the town, especially certain streets in that part, have had a bad reputation.

Under these conditions it may be of interest to investigate where the bad homes are located, and where the delinquent children and juvenile criminals have their homes.

From Table 39 it is evident that the distribution of the present material among the various parishes of the town deviates

Table 39. **Distribution of the cases on different parishes compared with the corresponding distribution of the total population of Malmö. Percentage figures.**

Parish	1903—1940					
	Group					Total population of Malmö
	I	II	III	IV	Total	
S:t Petri	7.8	12.4	11.4	12.0	11.0	16.0
Caroli	43.2	34.8	21.1	19.1	26.4	15.5
S:t Pauli	24.9	20.6	27.5	27.6	26.3	28.9
S:t Johannes ...	14.3	22.3	22.3	19.7	19.8	19.7
V. Skrävlinge ...	6.3	5.2	7.8	11.2	8.3	10.3
Limhamns	3.1	4.7	6.6	6.9	5.8	7.3
Fosie	0.4	—	2.8	2.8	2.0	1.7
Husie	—	—	0.5	0.7	0.4	0.6
Total	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	477	233	831	722	2263	—

strinkingly from that of the total population only as far as 2 parishes are concerned — Saint Petri and Caroli, especially the latter. The number of children taken into the charge has been lower in the former parish and higher in the latter than was to be expected from the relative total population of these parishes. Thus during the entire period, 1903—1940, about one-fourth of the boys and one-third of the girls came from the Caroli parish, while during the same period only about one-seventh of the population of the town resided in that parish. The differences are statistically significant for both boys and girls.

In reality, however, is a very great social difference between the Petri and Caroli parishes. Even though squalid dwellings are found in the Petri parish — especially the Malmöhus emergency rows — this parish is nevertheless to be considered as an upper-class parish, where as the Caroli is populated preponderantly by the poor classes. In consideration of this point it will be of interest to compare the distribution of the groups of the present material among the different parishes.

Just as before we find above all a marked difference between

the Caroli and the Petri parishes. In our material the Petri parish is less frequently represented. This applies above all to Group I (bad homes). In contrast thereto, the Caroli parish shows a considerable increase in the frequency of Group I as well as Group II, children from bad homes, but no significant change in the frequency of children from the other groups.

The compilations here presented are of interest in so far as they show that the social status of the parents determines to some extent which children are taken in charge. The findings here are in keeping with the conditions previously demonstrated in this material.

Of course it would have been more correct to employ for comparison the number of children in the various parishes, not — as done here — the total number of persons entered in the registers of the church.

The map, furnished at the end of the book, gives a survey of the distribution of the homes over different regiones of the town and shows a tendency to greater frequency in some parts.

CHAPTER IX.

FREQUENCY OF IMMIGRANTS IN MALMÖ IN THIS MATERIAL.

It may be of interest to see to what extent the individuals in the present material are natives of Malmö or immigrants.[1] A priori it can be assumed that the latter constitute a heterogeneous group of desirable as well as undesirable elements added to the population of the town. It is commonly believed that those who move into the towns are generally energetic and enterprising, while the slack and tardy remain in the villages. But, as far as I know, no scientific investigation has lent support to such an assumption.

It would seem safe to reckon, however, that a section of the immigrants is made up of unbalanced individuals, shy of work, who sooner or later become a burden to the community and, in so far as they bring a family with them, the children will have to be taken in charge by the Child-Welfare Board because of bad home conditions.

It is even conceivable that some immigrants are defective genetically, and that their children are particularly liable to become delinquent. But the children may also become delinquent because the milieu is unwonted, and provides stronger temptations.

Here it is to be mentioned that, of course, not all the immigrants in Malmö come from rural districts; many come from other towns. In the present investigation no attempt is made

[1] In this book, the term »immigrant» applies to all those inhabitants of the towns who are not natives, regardless whether they come from rural districts or from other towns.

at a division of the material in this respect. The number of individuals is too small to make such a classification worth while.

Some authors have tried to analyse the qualities of the immigrants in various ways. As these investigations are not reviewed in the survey of the literature, they will be mentioned briefly here.

DAHLBERG & STENBERG (1934) investigated whether the risk of alcoholism was distributed equally among the native and immigrant inhabitants of Stockholm. The outcome of this was that in two periods, before and after the year of the census, 1920 -- that is, in 1916—1920 and 1920—1925 — the number of persons reported to the Board of Temperance gave a decidedly higher percentage of natives than was to be expected considering the composition of the population of Stockholm. The authors think that this result is due to the fact that incipient alcoholics cannot immigrate at quite the same rate as other people.

DAHLBERG (1941) later investigated the risk of alcoholism in Malmö, and found it to be about the same for the natives as for the immigrants in this town. So it cannot be assumed that the immigrants to Malmö were an inferior group in this respect.

INGHE (1942) has looked into the distribution of immigrants and natives in a material consisting of young women who were hospitalized in Stockholm on account of induced abortion during the period of 1930—1934. Only 22.2 % of these women were born in Stockholm as against the total 32.1 % native women in the entire population of the town within the age-class of 15—45 years. Thus the author was able to demonstrate that a majority of the aborting young women in Stockholm were immigrants Furthermore, a special investigation showed that most of these immigrants had been in Stockholm for more than one year. So it was out of the question that this group of women hade gone to the city only for the sake of abortion.

FREY (1944) has analyzed a series of 600 young girls, 15—20 years old, who during the period of 1931—1941 had been reported to the Advisory Bureau of the Child Welfare Board in Stockholm, and found that more than half of the girls had

come from rural districts. No comparative figure is given for the total number of immigrated girls in that age-group. Of girls who could certainly be classed as prostitutes, no less than 36 out of 40 were immigrants. This investigation further showed that 1.3 % of all the immigrated girls in this age-class within one year after their arrival ran a risk of becoming liable to legal proceedings.

Subsequent investigations are of interest because they show how complicated these problems are. When country girls who move to town get into trouble, this may indeed sometimes be due to an unfavourable hereditary constitution such as lack of balance, etc., but it may also be due to an inability to get along under the new conditions. Therefore the given figures allow of no definite conclusion.

Where delinquency of children is concerned the significance of the change, in the external milieu differs widely, depending on the age of the child at the time of immigration. In very early childhood, no doubt, it plays a minor role. Small children soon get used to the new surroundings and accept them. But such a transfer to a new milieu is by no means a simple matter to school-children and, above all, to youngsters who move to town after leaving school. Other customs and ways of thinking, other ways of behavior and, in particular, more temptations meet them in the new surroundings and may bring about a radical change in their conception of things and their standard of conduct. All kinds of difficult situations may arise, especially for those young persons who, without the support of a home, move to town partly in search of better working conditions but, naturally, also driven by their longing for adventure and craving for amusement.

From Tables 40 and 41 it is evident that during the last two periods, 1916—1925 and 1926—1940, for which comparable figures could be obtained, a disproportionally high percentage of the »child-welfare material» was made up of children who were not born in Malmö, *i.e.,* immigrants. The difference between this percentage and the ratio of immigrants for the total population during the first period, 1916—1925, was most pronounced for boys of 10—15 years, being statistically significant

Table 40. **Percentage number of the cases who have moved into Malmö, distributed according to age and sex.**

Age when taken care of		Boys				Girls			
		Group							
		I	II	III	IV	I	II	III	IV
1903—1915									
0— 5		7.4	—	—	—	31.8	—	—	—
5—10		29.1	40.7	46.4	—	35.7	28.6	25.0	—
10—15		32.4	41.2	34.4	100.0	21.1	40.0	50.0	—
15—20		—	—	—	33.3	—	—	—	55.6
0—20	Per cent	25.2	41.0	35.9	34.0	29.9	37.5	41.2	55.6
	Total No.	119	61	181	106	117	32	34	9
1916—1925									
0— 5		25.0	—	—	—	—	—	—	—
5—10		22.2	25.0	15.4	—	25.0	14.3	33.3	—
10—15		35.7	28.1	31.2	—	31.3	18.2	31.3	—
15—20		—	—	33.3	26.3	—	—	—	23.8
0—20	Per cent	26.3	27.3	29.3	26.3	24.6	15.0	30.2	23.8
	Total No.	57	44	215	217	61	20	53	21
1926—1940									
0— 5		15.4	—	—	—	13.0	—	—	—
5—10		50.0	33.3	45.5	—	46.4	33.3	—	—
10—15		31.8	50.0	30.5	—	33.3	23.5	27.8	—
15—20		20.0	20.0	30.7	29.9	—	33.3	46.3	30.4
0—20	Per cent	29.0	42.6	31.3	29.9	31.8	26.1	41.9	30.4
	Total No.	69	61	227	367	66	23	160	23
1903—1940									
0—20	Per cent	26.5	38.0	31.9	29.3	29.1	28.0	39.3	32.1
	Total No.	245[1]	166	623	690[2]	244	75	247	53

[1] One case with unknown birthplace.

[2] Two cases with unknown birthplace.

for this group. During the latter period, 1926—1940, the same tendency asserts itself to an even greater extent, and is then specially pronounced in the youngest age-group, 5—10 years, being statistically significant for both boys and girls. For boys of 10—15 years the difference remains significant, but not for girls in the same age-class.

Table 41. **Percentage number of the cases who have moved into Malmö during different periods of time compared with the corresponding number among the total population of Malmö.**

$D \pm \varepsilon(D)$ = Difference ± standard error of the diff.

Age, years	Observed 1916—25	Malmö 31/12 1920	Difference $D \pm \varepsilon(D)$	Observed 1926—40	Malmö 31/12 1930	Difference $D \pm \varepsilon(D)$
			Boys			
5—10	20.0	17.2	2.8 ± 5.0	42.2	20.2	22.0 ± 6.0
10—15	31.0	21.0	10.0 ± 3.0	34.2	19.1	15.1 ± 2.9
15—20	26.4	31.9	5.5 ± 3.1	29.8	23.4	6.4 ± 2.0
			Girls			
5—10	23.3	17.1	6.2 ± 6.9	43.8	19.0	24.8 ± 6.9
10—15	29.7	21.9	7.8 ± 4.8	27.9	22.4	5.5 ± 5.1
15—20	20.0	38.0	18.0 ± 8.7	43.6	31.9	11.7 ± 3.8
			Both sexes			
5—10	21.1	17.1	4.0 ± 4.2	42.9	19.6	23.3 ± 4.5
10—15	30.7	21.5	9.2 ± 2.6	32.6	20.7	11.9 ± 2.5
15—20	25.7	35.2	9.5 ± 3.0	33.2	28.0	5.2 ± 1.8

It is perhaps to be mentioned first that in the periods prior to 1926, with few exceptions, the age group of 15—20 years is made up entirely of court cases, as before this year the upper age limit for intervention by the Child-Welfare Boards was 15 years. In the later period, 1926—1940, this age-group is made up of »welfare cases» as well as court cases. An analysis of this age-group reveals a divergence between the two periods. In 1916—1925 it is plainly evident that only a minority of the sentenced young delinquents were immigrants. Later, rather, the reverse was the case. In 1926—1940 a relatively great part of this age-group consisted of immigrants; and this applies to both girls and boys.

On the whole, then, this investigation may be said to show that a relatively high percentage of the »child-welfare» material is made up of immigrant children. Over $^1/_4$ of the children who are taken in charge on account of bad home conditions alone, and more than $^1/_3$ of those who are taken in charge on mixed

indication, come from immigrant families. The same applies to delinquents and court cases. On an average there are more immigrants among the girls in this material than among the boys.

Finally, attention should be called to the tendency which is to be seen among the girls in the group of serious delinquents in the last period. For, of the girls between 15 and 20 years of age nearly one-half were immigrants. Since 1934, the total number of girls in this age-group who have come under public supervision on account of licentiousness or indolence (§ 22 *d*) has been 28 — and one-half of them are likewise immigrants.

CHAPTER X.

STATE OF HEALTH OF THE CHILDREN.

As appears from the survey of the literature, delinquent children have sometimes been found to have poorer physical health than normal children, and the suggestion has been raised whether this might not in some cases contribute to delinquency. It is therefore interesting to see how this checks up with the present material.

It must be said at once that data as to the state of health of the children and juveniles is defective in several respects. Only in recent years has a systematic medical examination been made of children sent to detention homes — i.e. after 1938, when the detention-homes organisation was centralised and rules as to examination of these children drawn up. As this material ends with 1940 inclusive, very few of the Child-Welfare clientele have received any thorough medical attention. As a rule the only ones examined were those giving reason to suspect some pathological change at the time they were taken in charge. Thus, diagnoses of existing diseases come from physicians, whereas clean bills of health are based merely on the reports of the personnel of the detention homes in question, or on the views of parents or foster-parents. And the criminal juveniles have received no systematic medical examination at all, unless they had been in a reformatory.

The data as to the physical health do not cover the entire material, but only 1 091 boys and 425 girls (63.2 and 68.7 per cent respectively), and generally apply to the condition when taken in charge. For the vast majority, the health was said to be good. 'General debility' has been reported for both sexes to varying per cents during the earlier period, and is found

more often during the later period — up to nearly 40 % among children taken in charge on account of bad homes (group I). The information is vague, however, and it is difficult to assess its implications and how far it is objective or merely an indication that the children gave the authorities the impression of being not very healthy. The reports may quite likely have no common denominator. The figures should therefore be taken with a certain reservation.

It is hardly probably that chronically diseased children are included in the material, since these need attention of another kind. It is also hardly feasible to see causes for delinquency in bodily illness. It is more likely that certain physical defects or abnormalities in appearance or manner, which expose the sufferer to the merciless scorn of his comrades, might lead to maladjustment, and to a desire for self-assertion in some way; in other words, delinquency may be an expression for over-compensated inferiority complex. A survey of the material from this point of view has yielded very meagre results, however.

Different deformities of more or less disabling kind, which may in some cases have promted the epithet of 'cripple', are found in 22 boys and 1 girl, or nearly 1 per cent of the material investigated. The diagnoses have been the aftermaths of poliomyelitis, congenital spastic paralysis, lameness, spondylitic and rickets deformities in the form of kyphosis.

Another 'physical defect' noted is impaired hearing in 14 boys and 1 girl, i.e. rather more than 0.6 per cent of the material. And 1 boy's sight was so bad that he was later admitted to an institute for the blind.

Wetting the bed varied among the boys in the different groups between 13—17 per cent: it was lowest in Group III and highest in Group I. Needless to say, this symptom was not taken into account for children under school age. The criminal boys yield particularly low figures in this respect (about 1 per cent), but this is probably due to inadequate data. The girls present the same symptom to a considerably smaller extent, i.e. 10—15 per cent.

An investigation was made in the spring of 1946 among the children of an elementary-school in Malmö for comparative

Table 42. **Frequency of »Bed-Wetters» (n) among school children.**

Class	Boys			Girls		
	Total No. = N	n	n in per cent of N	Total No. = N	n	n in per cent of N
Normal.....	595	44	7.4 ± 1.07	578	33	5.7 ± 0.96
Weak (B-class) ...	110	6	5.5 ± 2.17	103	8	7.8 ± 2.64
Special (mental)...	105	20	19.0 ± 3.83	73	6	8.2 ± 3.21
Total	810	70	8.6 ± 0.99	754	47	6.2 ± 0.88

material. It was found that of the total number of boys (810), 70 (8.6 ± 0.99 per cent) wet their beds; the figures for the girls were 47 of 754 (6.2 ± 0.88 per cent).

The frequency seems to be higher among the boys than the girls, but the difference is not statistically significant. And we find higher figures for children in special classes than for children in normal classes, specially among the boys. The difference is 11.6 ± 4.0 per cent and is thus probable but not significant. For the girls the corresponding difference is 2.5 ± 3.4 per cent.

Compared with these figures, then, it seems as though bed-wetting was rather more common among the boys in this material than among other schoolboys, whereas the girls in this material show no higher frequency than normal, non-delinquent girls.

Other neurotic symptoms, such as stammering, have been registered for 11 boys (0.6 per cent), but not for any of the girls. Hereditary syphilis was found among 5 boys (0.3 per cent) and 11 girls (1.8 per cent); 31 girls (5.0 per cent) had been infected with gonorrhoea from sexual intercourse. The latter, however, is an effect rather than a cause of delinquency.

The diseases to which greater importance has been ascribed in the present instance are chorea, epilepsy and the sequels of encephalitis. Chorea has only been found in 3 boys (0.2 per cent) and 1 girl (0.2 per cent), while epilepsy was present in 6 youths (0.3 per cent) and 1 girl (0.2 per cent). Remains of encephalitis were found in 4 boys (0.2 per cent).

Another phenomenon thought to have some bearing on both criminality and delinquency is the presence of cranial trauma. In this material, this was said to have been present in 24 cases in all, 22 boys and 2 girls; this, calculated on the total number of cases, makes about 1 per cent of the number of cases on which there was any information. In 6 cases, no details were given; in the remainder, the injury was more exactly specified. Most of the injuries were the result of falls from heights, or road accidents. The children had either been treated in hospital, or stated to have lain unconscious for some hours after the injury, or to have shown other signs of cerebral lesion, such as vomiting or obstinate headaches. The age at the accident varied between 8 months and 15 years.

To assess the bearing of the injury on delinquency, it is naturally important to know whether any changes in disposition were noticed after it had been inflicted. In 2 cases it was stated that, after the injury, the boy had 'not been like other children'. There are no entries for the other cases as to the child's mental state before and after the injury. In half of these cases the injury occurred at so early an age (before 6 years) that the children would hardly have been able to demonstrate any delinquent tendencies before that time.

There are, of course, no figures illustrating how often serious head injuries occur in children in general. The frequency figure evolved in this material is consequently difficult to evaluate. It is probable that non-delinquent children are equally liable to injuries of the kind. In any case, head injuries are far too uncommon to be said to constitute any major cause of delinquency. It may be mentioned that, in all but one case, the children concerned were either those taken in charge for mild or grave delinquency, or else criminal juveniles. The one exception was a girl who had been taken in charge for prophylactic ends (Group I).

No. $^{4}/_{18}$ I This girl was taken in charge at the age of 3 years, together with 4 sibs, on account of bad home conditions, and was placed in a private home in the country. She was considered backward, but there is an entry to the effect that 'during her schooldays she showed no signs of delinquency'. At the age of 7 she just escaped a nasty accident, in which her

forehead was badly skinned. At the age of 15 she was given work in the foster-home. Some months later she set fire to a barn, which was burnt to the ground. The motive she gave was revenge on her foster-mother, who used to 'nag' her. In the time immediately preceding this, the girl had been difficult and moody, and often had migraine-like headaches. On investigation at the Psychiatry Clinc in Lund, an intelligence level of 9.5 years was established. Diagnosis: Dementia praecox, but epilepsy not out of the question.

Summing up, it can be said of the children in this material that their physical equipment was, on the whole, the same as that of children in general; if anything, the percentage of sickly subjects was low, though this of course may be mainly due to the somewhat inadequate data.

CHAPTER XI.

MENTAL CHARACTER OF THE CHILDREN.

According to a number of authors (e.g. WIMMER, GRUHLE, REHM, GREGOR, GLUECK and GLUECK), it has been asserted that delinquent children are somewhat lacking in intelligence. However, as even a stupid action requires a certain measure of intelligence, it goes without saying that major defects of intelligence such as imbecility and idiocy can hardly be expected among the delinquency clientele; we would rather expect it to be recruited from those of weak intellect and, in particular, from special-class children.

It would therefore be interesting to try to get an idea of the mental resources of the children included in this material. It must be said in advance, however, that great difficulties must be encountered and certain sources of error prove unavoidable in attempts of this kind on a material such as this one, which was not originally intended for investigation; the results reached must therefore be assessed with caution.

INTELLECTUAL EQUIPMENT.

There is data as to intellectual equipment for 1 374 boys and 475 girls, making a total of 1 849 children or 78.8 per cent of the entire material. Only a small number (106 or 4.5 per cent) of the investigated material have been tested. Most of the information consists of direct statements from detention homes and reformatories, and of teachers' opinion in the form of school reports or other pronouncements. Together, these latter categories make up rather more than $^4/_5$ of the information. Other sources of information were derived from statements as to whether the subject in question had been in a special class

(131 cases 5.6 per cent), or if he or she had left school prematurely on account of 'defective powers of comprehension' (56 cases=2.4 per cent) according to § 48 in the elementary-school statutes. This epithet, which is also used for children holding a certificate of discharge from special classes, can be taken as a completely sure indication that the child was considered to be intellectually lacking. Data on this head come from the time when there were not very many special classes, so that they complement the material from the earlier period in this point. If the only information offered is that the child did not get its remove, the child is classified as 'unknown', since failure to move up need not necessarily be due to backwardness. Except as regards the children subsequently transferred to detention homes, most of the data for the children from Group I (taken in charge due to bad homes) come from entries in the inspectors' case-book, since this contains regular notes both as to the health of the children and as to their behaviour in foster-home and school; most often they also contain remarks on the nature of the school reports. Naturally, the data apply not to the capacity of the children on the occasion of their being taken in charge, but to what it was later, when the child is old enough for its intellectual achievements to be assessed.

The information is of limited value since it comes from several different quarters. The verdicts which may perhaps be considered most reliable are, of course, the results of the intelligence tests, but then, these cover little more than 5 per cent of the material investigated. As previously pointed out in the survey of the literature, the course of an intelligence test may also be influenced by sources of error, which may be even greater in questions of delinquent children than when normal children are concerned. Delinquent children may very well prove recalcitrant in this respect, too, and accomplish less than their intellects are actually capable of.

Next in assessment are data as to whether the child in question has attended a special class. These classes were introduced in Malmö at an early date, the first class as early as 1900, but for several years they accepted only the more pro-

nounced cases with mental defects, mainly the imbecile and very retarded. Not until 1924—25 was a systematic testing for special classes introduced in connection with the expansion of these classes in the Malmö elementary schools. Thus, these children too — at any rate those from the later period — have been tested, although the intelligence quota is not given. To the assumption that these children are unquestionably subnormal, the plea might be raised that delicate children, or such as suffer from bodily disease and are therefore capable of less, might manage to pass unnoticed into these classes. This source of error can be more or less entirely eliminated, since all children recommended for a special class are first examined by the shool doctor.

Probably the most uncertain indicators of intellect are the school reports. After all, these have been given by different teachers, and consequently according to different principles of assessment. It is obvious that the personal opinion of a teacher must play a certain part, and that children who are in disfavour as a result of bad behaviour run the risk of a worse report not only for conduct but also as regards knowledge. It is also conceivable that delinquent children deliberately neglect their school tasks, and are further led by their special temperament not to attend to the instruction and to relapse into apathy and laziness, which means they get bad reports.

The estimation of the children's intellectual equipment is based direct on their reports in 509 cases (21.7 per cent). In only rather more than half of these the author herself has been able to calculate the total value. In the other cases, the respective investigator made pronouncements such as 'a pass', 'excellent marks', or else 'weak' or 'poor' marks, or 'just scraped through'. Thus, a certain subjective source of error cannot be avoided in calculation. It must also make itself felt in direct statements from the teachers in question, who must always be heard before the Child-Welfare Board makes a decision. These statements have sometimes been fairly noncommittal, such as 'not without talent', 'some talent', or 'difficult to teach', which has of course made the assessment more difficult. However, the reliability of the data is substantiated by the fact that those

from whom it came had been able over shorter or longer periods to follow the progress of the subject; this, of course, also applies to data from the personnel from detention homes, which data was provided in 543 cases. These latter data also come from teachers, who have a certain practice in assessing a child's general mental standard, so that they can safely be used. According to NORDAL's investigation (1935), there was quite good agreement between the judgments of the teachers and the tests he himself carried out. He submitted a deviation from the teachers' statements of 13 per cent in one investigation, and of 7.3 per cent in another. However, he does not say whether these statements were too favourable or the reverse.

On the guidance of the data obtained, the material has then been divided up into different categories: good, medium and weak intellect, and feeble-minded (debile) and imbecile. In the cases where a test was made, an I.Q. of 90—100 has been reckoned as moderate. 100 and over was good, between 80—90 weak, and 70—80 feeble-minded; both these last figures give the levels used in the Malmö elementary schools for transference of children to »weak-class» and special class respectively. In cases where school reports were accessible, children with an average mark of 1—1 $^1/_2$ were reckoned of moderate intellect, while 2—2 $^1/_2$ marks was accounted good. $^1/_2$—1 mark was weak; this also included those belonging to the weak-class, also called B-class. Statements from the teacher such as 'not ungifted', 'some talent' have led to the subject's being placed in the category 'moderate intellect', while expressions such as 'very slow in the uptake', 'difficult to teach' were taken to indicate weak intellect. Children denoted 'retarded' or 'very retarded' were accounted feeble-minded.

Children relegated to this last group, then, were those coming through the tests with an I.Q. of 70—80 and those labelled 'retarded' by the teachers, but even more those reported to have belonged to a special class, and, finally, those who had left school prematurely on account of 'deficient powers of comprehension' (§ 48). As mentioned already, these children are to the earlier period what the special-class children are to the later one. Both these two categories have also been

described separately, to make possible a comparison with the frequency of such children generally.

This distribution, too, must inevitably be based on subjective assessments. It is not always easy to decide when a verdict justifies the placing of a subject high on the intellectual scale, or whether he shall go down a step. Difficult and uncertain as it often is to determine between weak and moderate intellect *in casu,* it is still worse when the decision is to be made according to often vaguely formulated judgments of others. As, however, the material has been judged uniformly through out, a comparison between its different groups is possible.

In the following calculation, the percentages have been calculated not only on the number where data exist, but also on the total number. It seems natural to assume that, if a child is particularly lacking or has belonged to a special class, or if it has particularly good marks, these points would be mentioned, and consequently entered in the records. On the other hand it is very likely that ordinary, normally gifted children are not entered as such, because such endowments are taken for granted. And a closer survey of those reckoned as 'unknown' shows that, in more than half of the cases, a note was made of the class to which they belonged, and in practically all these cases, the age of the child corresponded to that of the class. There should therefore be every reason for supposing that these children are normally gifted. However, in order to keep to as reliable data as possible, the author has not included these cases direct in the number of 'normally gifted' children.

If we start with a total survey of the material, we find that 8 cases, 5 boys and 3 girls, have been denoted imbecile. Of these, 4 belong to Group I (children taken in charge on account of bad homes), and it is quite natural that children of so low intellects might be included here, since many were taken in hand so young that no pronouncement as to their intellectual capacity was possible. The remainder belong to the two Groups II and III (mild and severe cases of delinquency), whereas none belongs to Group IV (criminal juveniles). Thus, the delinquency clientele proper has come to include 2 boys and 2 girls whose manifestions of delinquency may well be symptoms of their

Table 43. **Percentage distribution of the children according to intelligence.**

Intelligence	1903—1940							
	Boys				Girls			
	Group							
	I	II	III	IV	I	II	III	IV
Good	22.4	21.1	15.9	21.8	27.9	17.3	21.1	13.2
Medium	26.8	27.7	36.8	37.8	19.7	38.8	29.1	37.7
Weak	15.9	21.7	16.2	13.6	10.2	17.3	19.0	13.2
Debile	8.9	10.2	8.7	8.9	7.4	9.3	17.4	5.7
of these special class children	3.7	8.4	7.7	8.8	3.7	9.3	15.0	3.8
Imbecile	1.2	0.6	0.2	—	0.4	1.3	0.4	—
Unknown	24.8	18.7	22.2	17.9	34.4	16.0	13.0	30.2
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of cases	246	166	623	692	244	75	247	53

low intellectual equipment rather than indications of real delinquency, and who ought to have been placed at once in other institutions. When the cases in Group I are subtracted, however, the number is extremely small, 0.1 and 0.4 per cent of the boys and girls respectively, so that they only deserve mention for the sake of completeness.

No. $^{7}/_{28}$. II. Of the two cases included in Group II (mild delinquency), the boy, who belonged to a well-known gypsy family, was taken in charge at the age of 8 on account of pilfering and inadequate home supervision. All his brothers and sisters had incurred intervention by the Board, and the home had been denoted 'the worst imaginable'. He belonged to the imbecile class at school with an I.Q. of 53, had a very bad temper, was extremely hard to bring up, and showed several symptoms of 'general delinquency' which could not be interpreted solely as symptoms of feebleminedness. He was first treated at a children's home, whence he came to a detention home and was transferred thence to an asylum, where he was kept 2 years on the diagnosis: *Imbecillitas*. The total period of treatment was 11 years and 4 months. Treatment at an ordinary institution for the feebleminded would not have been possible for his boy.

No. $^{8}/_{08}$. II. The girl belonging to the same group was taken in charge by the Poor Relief Board at the age of 12 years, because she was an orphan. She was placed in several homes, but no foster-home would keep her. On the grounds of 'weak intellect', 'unreliability and low moral standard' she was subsequently taken in charge by the Child-Welfare Board and

placed in a detention home, but only spent 10 months there, after which she was transferred as imbecile to the care of a private family.

No. $^{17}/_{06}$. III. Boy in Group III; taken in charge at the age of 9 years. Only reason noted was that he was 'highly delinquent'. He was taken in charge of the Child-Welfare Board for 5 years, of which 3 were spent in a detention home, and was subsequently taken in hand by the Poor Relief Board. Later entries showed that he was obviously profoundly imbecile, unable even to take care of himself in regard to feeding and clothing. Death at the age of 32 years from a foreign body in the oesophagus.

No. $^{3}/_{30}$. III. Girl in Group III taken in charge at the age of 17 years on account of extremely loose living. It was impossible to get her to keep to a regular job; she was away from home several weeks at a time, and hung about the boats in the harbour. On investigation she was found to be imbecile, and she was transferred shortly afterwards to the appropriate institution.

If the different groups are then inspected separately, we might expect children belonging to group I, who had been taken in charge solely on account of bad homes, to have approximately the same mental equipment as people in general, and to be able to serve as a control for the rest of the material. We find, however, that 24.8 per cent (33.0) of the boys [1] are subnormally endowed. Leaving out of account all but those who show more pronounced lack of intellect, i.e. the feeble-minded, we find these with a weak intellect to make up almost 9 (11.9) per cent. Of these 4 (4.9) per cent have attended a special class. This figure corresponds fairly well with DAHLBERG's (1937) percentage for the number of schoolboys in Stockholm who belonged to special classes, namely 3 per cent. This shows that the proportion of boys in this group who attended a special class is hardly larger than that found among children in the total population. It must, however, be remembered that a large number of these boys had been placed out in the country, where there are no special classes, and that the most reliable figure is probably that of the total frequency of feeble-minded cases, which as mentioned is 9 (11.9) per cent. It is probable, then, that the frequency of boys in this group who are ripe for special classes is 3 times higher than among the children of the normal population.

The category 'weak' intellect will always constitute an inter-

[1] Henceforth all percentages imply calculations on the total number, and the figures in brackets calculations on the number with data.

mediate class of indefinite character. The boys in Group I belong there to about 16 (20) per cent, thus corresponding to so-called physiological stupidity, for which there are no frequency figures among the normal population but which may well be considerably higher there. Normally gifted boys in this group make up 49.2 (65.4) per cent of it. If 'the unknown' are also accounted of normal intellect, then 74.0 per cent, or about $^3/_4$, would be normally equipped, whereas $^1/_4$ are lacking to greater or lesser degrees.

Of the girls in this group 3.7 (5.6) per cent have attended a special class. This figure is nearly twice that of DAHLBERG's normal figure for girls: 1.7 per cent. The total number of feeble-minded cases constitute 7.4 (11.3) per cent; that is to say, more than 4 times more girls in this group than among normal children have an intellectual capacity entitling them to admission into a special class; 10 (15.6) per cent have weak intellects, while 47.6 (72.5) per cent are said to be, and probably 82 per cent actually are, more or less normally gifted (normal intellects+unknown). According to this latter calculation it seems as if the girls in this group are rather better equipped than the boys, and this also comes out if the percentual calculation is made only on the number about whom there is any information (72.5 per cent for girls as against 65.4 per cent for boys).

In Group II (children taken in charge on mixed indications, i.e. mild delinquency and bad home conditions), the frequency of feeble-minded boys is 10.2 (12.6) per cent, that is to say, at least 3 and in all probability 4 times higher than the accepted norm. Of these 8 (10.4) per cent had attended a special class. The entire number of subnormal boys constitutes about 32 (40) per cent, while not quite half are normally gifted, which if the unknown are added makes 68 per cent.

Rather more girls than boys have attended special classes in this group — 9.3 per cent as against 8.4 per cent; but the difference is small. Compared with the normal figure i.e. 1.7 per cent for girls this means, however, that feeble-minded girls are in this group 6 times as many as usual. The frequency of girls with weak intellects is 17.3 (20.6) per cent, not quite

so high as among the boys; the frequency of normally equipped subjects also shows the girls to be better off, with 56.1 per cent against 48.8 per cent of the total number (66.7 as against 60.0 per cent) for the boys.

Among the cases of more grave delinquency (Group III), 8.7 (11.1) per cent of the boys have been registered as feeble-minded. Of these 7.7 (9.9) per cent have attended a special class. Compared with DAHLBERG's figure of 3 per cent, 3 times as many of these boys as boys in general have so low an intellectual capacity that they can be considered as 'ripe for the special class'. But over half the number, 52.7 (67.9) per cent, are unquestionably of normal intelligence. If we continue to suppose that those for whom there are no data are 'normal', then $^3/_4$ of the delinquent boys can be considered of normal intelligence. If to this we add the category 'weak intellect', which can be considered to represent 'physiological stupidity', we find that about 90 per cent are no stupider than most people.

The frequency of feeble-minded girls is high in this group, however, not less than 17.4 (20.0) per cent. Of these 15 (17.2) per cent have attended a special class, which on comparison with DAHLBERG's earlier mentioned normal figure (1.7 per cent) makes 10 times the number found among girls generally. Of the total number of girls 36.4 (41.9) per cent are subnormal — i.e. more than every third delinquent girls shows defects of intelligence to a greater or lesser degree. The frequency of normally gifted girls in this group is 50.2 (57.6) per cent, and if the unknown are added to this figure, we get 63 per cent.

In this group more girls are retarded than the boys in the same group. Even if the difference in weak intellects is small, the number of feeble-minded are appreciably higher among the girls. This difference is 8.7 ± 2.7 per cent and is significant. Thus, extremely lowered intellectual powers are twice as common among delinquent girls as among delinquent boys.

Of the criminal juveniles (Group IV), 8.9 (10.9) per cent are feeble-minded, and practically all have attended a special class. A comparison with the normal figures shows special-class boys to be 3 times as frequent here as among boys in the elementary schools. If cases of weak intellects and feeble-minded are put

together, we get the figure 22.5 (27.4) per cent — that is to say, at least every 5th boy shows at all events a slight defect of understanding. Direct records of normal intellect yield the figure of 59.6 (72.6) per cent which is a minimum number, while the maximum number, normal+unknown together — amounts to 77.5 per cent; that is to say, in at least more than $^1/_2$ and perhaps $^3/_4$ of this clientele, the criminal conduct cannot be imputed to faulty intellectual development.

Of the girls in this group 5.7 (8.1) per cent are feeble-minded, and in these about 4 (5.4) per cent have attended special classes. At least half of the group, and probably more than 80 per cent, are certainly of normal intellect, and the remaining have sub-normal intellects. The number is too small to warrant any pronouncement, however. Nevertheless, if this small number of girls is compared with the boys in the same group, the intellectual level seems to be about equal. If this group, who has been more directly addicted to criminal ways, is then compared with the group of delinquent girls in Group III, it seems as though the former were more intelligent than the latter, where sexual offences are the dominating cause of intervention. The difference between feeble-minded girls in the two groups is 11.7 ± 4.0 per cent, and is thus probable.

A comparison between the different groups shows that about 8 (10) per cent of the boys in Groups II, III and IV attended special classes. There is only half this percent in Group I, however, though this may well be because most of these children were placed out in the country where there were no special classes. If the total frequency of feeble-minded is compared — i.e. including those children who, according to other sources of information, were to be regardad as ripe for a special class — this difference is smoothed out, and we get about 9 (12) per cent of feeble-minded boys in all groups.

The frequency of weak intellect is also at about the same level for the different groups. In round numbers $^1/_6$ of the boys come under this head. This figure is nearly $^1/_5$ for the boys in Group II, but this may well be due to chance.

With regard only to direct information, we find that scarcely half the boys in Groups I and II had normal intellects, while

Group III has more than half, and Group IV even more than this. Thus, even if the frequency of feeble-minded cases is about the same in all groups, and not smaller in Group IV, this latter group nevertheless leads by some distance, in that the number of weak intellects is less and the number of average intellects rises.

As seen in Chapter IV, many of these latter youths came from good homes. But, apparently, neither better environment nor better understanding have been able to keep them from crime.

A comparison between the girls in the different groups shows that the frequency of those attending special classes is lowest in Group I and Group IV. For Group I, probably for the same reason as held for the boys in the same group, this figure is too low and the lowest frequency of girls in special classes is found in Group IV, but this group is so small (53 cases) that the percentage figures are uncertain. The highest frequency of feeble-minded is in Group III (delinquent girls). The frequency of girls with weak intellects is also high in these two Groups II and III (mild and more serious cases of delinquency). It therefore seems as though the delinquent girls in this material have the worst intellectual standard.

Finally, let us compare the intellectual equipment of boys and girls. The frequency of the subnormal, which covers weak intellects and feeble-mindedness, is 24.5 (30.9) per cent and 26.3 (34.3) per cent for boys and girls respectively. The frequency of normal intellects, here designated as good and moderate intellect, is 54.7 (68.8) per cent for boys and 49.8 (65.8) per cent for girls. If, as before, we assume normal intellects for all those about whom there is no data, we find that 75.5 per cent of the boys and 73.7 per cent of the girls (i.e. $^3/_4$) are normally endowed. The difference is not significant, however, and may be due to chance.

In the population, stupidity seems to be more widespread among boys than among girls. According to the official statistics the frequency of imbeciles is higher among boys. Similarly, the frequency of special-class children is, according to DAHLBERG (1937), twice as high among boys as girls (3.0 as against

1.7 per cent). This does not apply to the children in this material where, as previously stated, the boys are on the whole more intelligent than are the girls. The difference is particularly marked in Group III (more serious cases of delinquency), where feeble-mindedness among the girls is especially high, and where the difference between the sexes in this respect is statistically significant. Now, in a normal population, the girls are less often subnormal than the boys, so that in this material the position has been reversed. We must see this in the light of the fact that delinquency is less usual in girls than in boys, and that girls need a stronger impetus from their environment to turn them delinquent. Under such circumstances it is not so surprising that lack of intellectual gifts sways the delinquency of girls more than that of boys.

A comparison of the intellectual equipment of the two different periods would certainly seem to show that the frequency of feeble-minded subjects was greater during the latter than during the former period, and that special-class children in particular were more numerous. However, this latter circumstance is an extremely natural result of the development of special classes, which did not become more common until the later period. The large number of feeble-minded during this later period may well be the result of better information, and anyhow it has not been considered necessary to make a separate survey of the two periods.

It is only as regards the girls in Group III (cases of more serious delinquency) that a marked difference comes out between the different periods. During the first period, when the girls were under 15 years old when taken in charge, the frequency of normal intellects is higher and that of feeble-minded cases considerably lower than during the later period. The difference is 21.6 ± 3.9 per cent and significant. This difference can probable be attributed wholly to the fact that, after 1925, older girls whose main offence was of a sexual nature, were added to this group, and that several of these — in particular after 1934 — belong to the category which may be called prostitutes. On the evidence of several investigations, comparatively

many of these girls had a particularly low intellectual standard (KEMP, NORDAL, BURT).

Summing up, then, we can say that the frequency of feeble-minded boys in this material is 3 times that for children in general. Except in Group III (more serious delinquency), where feeble-mindedness is 10 times the normal figure, the girls yield approximately the same figure. In round numbers, we can say that about 10 per cent of the cases exhibits extreme defects of intelligence, and this applies whether we calculate on the total number of children or only on those about whom there is any information. If, however, we now add the frequency of those with weak intellects, we find that only about $^1/_5$—$^1/_4$ of the total are lacking, and roughly between $^3/_4$ and $^4/_5$ are fairly normally endowed. Even if the frequency of lowered powers of intellect is higher among delinquent children than among normal ones, this number still only constitutes a minor part of the category of delinquents. It is important to be able to establish that children blessed with normal intelligence may become delinquent. In other words, normal intelligence is not enough to keep delinquency away.

FREQUENCY OF TEMPERAMENTAL ABNORMALITIES AMONG THE CHILDREN.

It is no easy task to estimate the frequency of »psychopathy» in this material on available information as to the character of the children. For one thing, the diagnosis »psychopathy» is itself vague and indefinite. For another, the verdicts passed are by no means easy to interprete. In a sense, delinquency can be taken to imply some abnormal trait of character which can always be called psychopathy if this concept is very broadly applied. Furthermore, only rather over half of the cases (1428) i.e. 60.9 per cent, are accompanied by any opinion as to peculiarities of temperament in children. A smaller number have been investigated by psychiatrists, and a limited number, 70 cases (53 boys and 17 girls) have been diagnosed as »psychopathic»; this was naturally more frequent in the later period than in the earlier one. The proportion of cases so diagnosed is therefore not quite 3 per cent of the whole

material or, counting only those children about whose psychological characteristics there was any evidence, less than 5 per cent.

On the other hand, if we take the collective heading »abnormalities of temperament» to cover various deviations from the normal standard of behavior, such as tantrums, bad temper, a tendency to destroy or cut up clothes and books, studied wickedness and cruelty, advanced powers of fabrication, and further such opinions as »not quite normal», »hard to bring up», we find several features indicating mental abnormality among 327 boys and 88 girls. Taking this together with the above-mentioned cases of psychopathy in the narrow sense we get 485 cases where emotions or will power had suffered, which constitutes 20.6 per cent of those cases concerning whom information is available. For boys and girls the percentage are 22 per cent and 16.9 per cent respectively, rather higher for boys than for girls. It is also found that pronounced untruthfulness is noted for 168 (15 per cent) boys and 26 (8.4 per cent) girls. It is therefore obvious that among these nearly 1 500 cases there are a number of individuals who have given proof of bad traits of character, although these figures must be taken as minimum ones.

Naturally, there are no data to the incidence of these traits of character among the population as a whole. The figures given must therefore speak for themselves. If the various groups are examined separately, it is found that the group of seriously delinquent children (Group III) shows the highest figures, and that more boys are affected than girls. Even in comparison with criminal boys these children show worse traits of character. Thus, psychopathy in the wide sense is evident in 100 out of 445 (22.5 per cent) delinquent boys, as against 58 out of 464 (12.5 per cent) criminal youths. The less good qualities, such a as laziness, untruthfulness and unreliability are also most in evidence among delinquent boys. However, in a way this is to be expected, as such characteristics are less searched for and less noted among the criminals than among the delinquents.

However, it may be worth noticing to what extent such temperamental traits in delinquent children are accompanied

by different degrees of intellectual ability. Previous investigations (e.g. WIMMER, v. GRABE) show that children with weak intellects are often psychopathic, but as far as I have been able to discover, there are no data as to how often such traits of temperament are found in children considered to be normally gifted; they might, perhaps be expected to show such traits more often, to explain their conduct.

If, then, we calculate the frequency of psychopathic features (in the wider sense) combined with various intellectual ability, we get the following: For the boys in Group I, 9.1 per cent (11 out of 121) of the normally gifted, 20.5 per cent (8 out of 39) of the weak intellects, and 45.5 per cent (10 out of 22) of the feeble-minded exhibited temperamental traits of the type mentioned above. If the two groups of delinquents (II and III) are made into one, we find that 25.9 per cent (106 out of 410) of those with normal intellects can be accounted psychopathics in the wide sense, whereas 53.5 per cent (38 out of 71) of the feeble-minded have the same disposition. The difference is 27.6 ± 6.3 per cent (significant). The cases considered to have weak intellects take an intermediate position with 34.3 per cent psychopathics (47 out of 137).

There are not quite so many entries of abnormal traits of temperament for the girls. Traits which could be accounted psychopathic symptoms were found in 7.8 and 8.0 per cent (9 out of 116 and 2 out of 25 respectively) among the normal and the weak intellects respectively in group I, in which only a small number of girls showed delinquency after being taken in charge, and can therefore be regarded as control cases. The feeble-minded, on the other hand, showed 28 per cent (5 of 18) psychopathics.

If, however, we include those girls showing delinquency of milder or more serious kind (Groups II and III), we find that, in round numbers, rather over 20 per cent showed abnormal traits of temperament. The difference between the different categories of intellect is not particularly large: 23 per cent (38 out of 166) of the normal intellects, 22 per cent (13 out of 60) of the weak intellects, and 26 per cent (13 out of 50) of the feeble-minded. The difference is not significant.

Thus, while there seems to be a certain relation between psychopathic features and weak intellect as far as the delinquent boys are concerned, no such connection is shown by the figures for the delinquent girls. However, the percentages for the girls should be assessed with caution, since they have been calculated on small absolute numbers.

The information given above concerning abnormal traits of character (psychopathy) is certainly incomplete and therefore of doubtful value.

The same is also true to some extent of cases of insanity. This is however rare in the earlier years of childhood and one does not really expect to find such cases before puberty. Most of these cases will have been investigated by doctors and put under supervision of another kind. In this respect therefore a community of delinquents is, to some extent, selected. There are however included in this material some cases which were originally interpreted simply as cases of delinquency but which later turned out to be cases of insanity in childhood. Altogether there were 7 such cases, 5 boys and 2 girls. The diagnosis obtained from the hospitals where they were admitted were schizophrenia in 5 cases and psychosis from infection (Encephalitis?) in 2 cases.

It may be added that 2 of the cases of schizophrenia were youths who were taken in charge at the age of 17 and 18 years in accordance with the regulation of 1934 concerning »an idle and vicious mode of life». Altogether this material only includes 8 youths taken in charge in accordance with this regulation, and 2 of these have not been cases of delinquency but insanity.

CHAPTER XII.

MEASURES TAKEN BY THE AUTHORITIES.

The main rule in the action taken by the Child-Welfare Board is that the child is removed from home and parents 'to be handed over to another for care and upbringing'. This means that the children are placed either in children's homes, in private homes or in detention homes. The direct reason of the Board's intervention is of a certain importance here, insofar as one must take into account whether the child was taken in charge as being delinquent or non-delinquent.

As regards the latter non-delinquent children, who in this material belong to Group I, the main action of the Board is to separate the children from their own homes and place them in private homes. During the first 12 years that the Board was functioning, which was before children's primary homes could be set up, both these children and those taken in charge for delinquency were at first placed in the alms-house, together with adults of both sexes, who often proved hardly suitable companions. In some cases, moreover, the time in the alms-house was relatively long, which may often have proved detrimental to the child's future development. After this 'quarantine', most of the children in Group I (170 boys and 171 girls, making 96 per cent) were, during the first period, placed in private homes, and only 14 children (8 boys and 6 girls, making 4 per cent) in children's homes. After the erection of a children's primary home in Malmö in 1915, the usual course was a preliminary placing there, followed after some period of observation by transference to a foster-home or children's home. During the later period, 42 children (25 boys and 17 girls, making 31 per cent) were placed out in children's homes, while 79

children (59 per cent) went to private homes. The former arrangement, less usual during the earlier period, has thus become more common during the later one. This holds even if the calculation is made according to the different length of the periods, 25 years for the former, and 15 for the latter.

Most of the private homes were situated in the Scanian countryside, not far from Malmö. This admittedly has its advantages from an administrative viewpoint, since it is easier for the Board to inspect and supervise the children, and possible troubles can more rapidly be seen to. But there are a number of drawbacks in placing the child so near the parent home. One of these was the more or less constant visits of the parents to the foster-homes, and various episodes quoted by the inspectors testify to the rather unhappy influence of these parents. Consequently the break with the old home has been by no means complete. As well as this, a number of the children in the first period were even allowed to remain in Malmö, though in other homes than those of their parents. Naturally the children visited the parents as often as they got the chance, and vice versa.

Sometimes these children had to be re-placed. Some of them proved more difficult to bring up than others, and new foster-homes had to be found, and sometimes the children had to be transferred to detention homes; this last occurred in 34 cases, 13 boys and 21 girls.

Needless to say, the placing of children taken in charge for delinquency has varied according to the nature and degree of the offence. As so few detention homes were available during the early period, more delinquent children were placed in private homes during this period than during the later one, when detention homes came more to the fore. The destination chosen has in all likelihood been governed to a certain extent by the fact that at an earlier date it was easier to procure foster-homes even for extremely delinquent boys. The difficulty of securing suitable foster-homes has increased in the course of time — a circumstance which must in itself increase the number of children placed in detention homes. During the first period, then, more than $^3/_4$ of the boys in Group II (mild cases of delinquency) could be placed in private homes, and

$^1/_4$ in detention homes after private homes had first been tried. During the later period, roundly speaking $^1/_3$ (21 cases) were placed in private homes, $^1/_3$ (19) in children's homes, while the rest (21) went after a period in a children's home or a private home to a detention home. Of the girls in the same Group (II), only rather more than half (30 cases) could be placed in private homes during the first period, while the rest landed up sooner or later in detention homes. During the later period the distribution was about the same as that for the boys, i.e. with $^1/_3$ in each of the 3 possible destinations.

The main rule in Group III (more serious cases of delinquency) is to place the children in detention homes. This measure was decided upon for 396 boys, but in 22 of these it was for some reason not carried out. Of the remaining 374, however, over half (193 cases, or 52 per cent) were placed in private homes during the first period, while 153 (41 per cent) went to a protective home, either direct or after some time in a private home or children's home. Added to these were 28 boys who had been admitted to the so-called *Internatet* (boarding-school), an institution which in certain ways was a forerunner to the detention homes, and which came into being in Malmö at the end of the last century, on the initiative of the elementary school board. This institution has of course to a certain extent served as a detention home and, on the completion of City of Malmö's own detention home, *Ö. Spång* (East Spång), was incorporated into the same. But even including Internatet as a detention home, only half the boys were placed in such homes during the early period, as against 75 per cent (165 of 220) during the later period, when about 15 per cent (34 cases) could be placed in private homes, and the rest (21 cases) in children's homes. The difference between the two periods for detention home treatment is 26.6 ± 3.9 per cent, and is statistically significant. As already mentioned, this cannot be taken to indicate an increase in the serious delinquency type, but must be assessed in the light of supply and demand in respect of detention homes.

It is noteworthy that rather more of the girls in the same group (III) were placed in detention homes during the first

Table 44. **Percentage distribution of the convicted cases according to measures imposed by the Court.**

First sentence	Boys		Girls	
	1903—25	1926—40	1903—25	1925—40
Probation	37.7	63.6	26.7	74.0
Fine	19.4	22.0	26.7	17.4
Reformatory education	14.5	10.3	36.6	4.3
Imprisonment	4.3	1.4	—	—
Penal servitude	24.1	1.9	10.0	—
Insane	—	0.8	—	4.3
Total	100.0	100.0	100.0	100.0
Number of cases	324	368	30	23

period: about 71 per cent (58 of 82) as against 50 per cent of the boys. The difference is not quite as great during the later period, when more than 91 per cent of the girls (142 of 156) were thus placed, against 75 per cent of the boys. The difference between boys and girls is respectively 22.3 ± 5.6 per cent and 16.0 ± 3.7 per cent during the two periods, and is significant for both.

The most immediate explanation may well be that the predominant type of delinquency among girls, i.e. sexual offences, is considered to be more difficult to overcome in private homes than in a detention home, since the latter can provide better supervision.

It may also be interesting to study the nature of the punishments to which the juveniles in Group IV were sentenced. A certain difference in the frequency of the different offences during the different periods can be noticed. Thus, during the first period, 37.7 per cent (122) of the boys were released on probation, while this was granted to 63.6 per cent (234) boys in the later period — i.e. $^2/_3$ as against $^1/_3$ in the first period. The difference is 25.9 ± 3.7 per cent, and is thus statistically significant. Sentences involving loss of liberty were passed in 28.0 per cent during the first period, but only in about 3 per cent in the later one. A survey of the nature of these sentences shows that 24 per cent (78) of the boys were sentenced to hard

labour during the first period, but only 1.9 per cent (7) during the later one. Imprisonment occurred in 4.3 per cent (14 boys) and 1.4 per cent (5 boys) in the earlier and later periods respectively. Fines were levied to about the same extent during both periods, i.e. 19 per cent and 22 per cent respectively. (Table 44.)

During the earlier period, 14.5 (47) per cent of boys and 10.3 per cent (38) during the later were sentenced to education in reformatories; thus, there is a difference here, too, though it is not statistically significant.

Among the girls, too, probation can be observed to occur more often during the later than during the earlier period (74.0 per cent as against 26.7 per cent). The difference is 47.3 ± 12.2 per cent. Rather more girls were sentenced to education in reformatories during the earlier period than during the later one. There are so few girls in this group, however, that a closer analysis could have no general importance. All the same, it is noteworthy that relatively more girls than boys have been committed both to reformatories and to detention homes.

Consequently, it is obvious that the punishments meted out during the earlier period were more severe than those in the latter, which might of course be taken to indicate that the crimes during the former period were of a blacker hue. Another glance at Table 23, p. 134 (grounds of prosecution) shows that this can hardly be the case. It is true that plain theft is more common during the later period, but the number of burglaries has risen. The later period also contains offences of a milder kind, such as illicit use of others' property, which usually means the 'borrowing' of cars. However, these are not enough to explain the difference, which is probably mainly due to altered practice.

LENGTH OF THE PERIOD UNDER CARE.

Needless to say, the length of the period under care has a certain connection both with the age at which the subject was taken in charge and with the laws in force at the time. To a certain extent, however, the child's own temperament affects the length of the care given, so that it will vary. A brief account

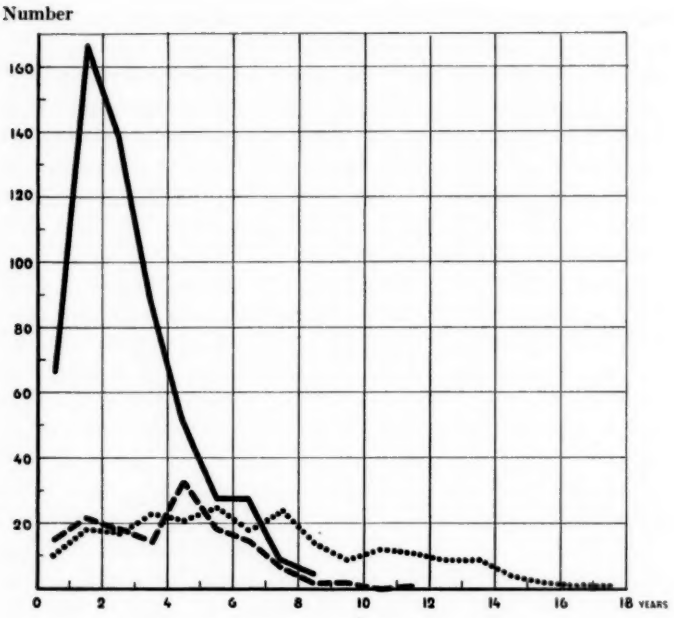


Fig. 8. Length of detention (in years) for boys during the period 1903—1940. Group I: dotted line; group II: broken line; group III: whole line.

of the length of this period in the present material may not, therefore, be out of place.

We may mention that by 'period under care' we mean only that period during which the subject was in charge of the Board — i.e. from the date of the move from the parental home to the date of discharge in probation. The period during which the subject was under supervision is not included.

There is no need to go into the reasons why Group I must have the longest period of care. Here the length of the care depends on the age when the subject is taken in charge: the younger the child, the longer should this period be. The law stipulates no maximum or minimum period for children taken in charge for prophylactic ends, but this time nevertheless has

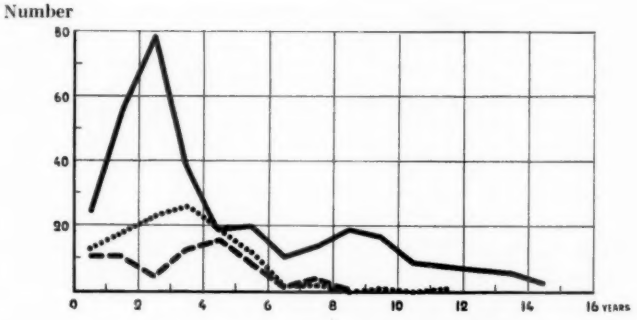


Fig. 9. Length of detention (in years) for girls during the period 1903—1940. Group I: dotted line; group II: broken line; group III: whole line.

something to do with the age at discharge as laid down by the law, i.e. 18 years. However, the length of the period may also be influenced by the fact that a change for the better has taken place in the parental home, and the parents demand the return of the child. Unless there are very solid reasons for retaining the children, they must then be returned to their parents. The 'improvement' in home conditions sometimes disappears after a shorter or longer time, and sometimes, indeed, is only superficial, anyway, so that the Board has to step in again. This naturally means that the children are in and out between children's home, foster-home and parental home. Discharges after a short period and readmissions of this kind have occurred in 20 cases during the later period (11 girls and 9 boys).

As Table 45 shows, the average period of treatment for the boys in this group was nearly 7 years during the earlier period, and not quite 6 years during the later one. This tendency to a shorter time during the later period cannot be due to a certain number of the children still being in charge of the Board, as such cases were not included. But on the other hand those included here are a selection of children with a short period of treatment. Nor can we say that the children were taken in charge at a later age during the second period than during

Table 45. **Mean time during which taken care of (in years) for the cases of the Child-Welfare Board.**

$M \pm \varepsilon(M)$ = mean ± standard error of the mean.

	Boys			Girls		
	Group					
	I	II	III	I	II	III
			1903—1925			
Number of cases	176	101	371	169	52	82
$M \pm \varepsilon(M)$	6.9 ± 0.55	3.8 ± 0.43	3.0 ± 0.19	6.4 ± 0.58	3.7 ± 0.58	3.4 ± 0.33
First quartile.....	4.0	1.8	1.5	3.2	1.8	2.0
Median	6.6	4.2	2.6	5.8	3.9	2.8
Third quartile ...	9.6	5.3	3.9	9.3	5.1	4.2
			1926—1940			
Number of cases	50	49	214	44	20	154
$M \pm \varepsilon(M)$	5.8 ± 1.3	4.2 ± 0.66	2.6 ± 0.22	4.9 ± 0.99	3.1 ± 0.79	2.4 ± 2.3
First quartile.....	2.1	2.8	1.4	2.5	1.1	1.6
Median	4.8	4.2	2.1	3.9	3.6	2.2
Third quartile ...	8.6	5.4	3.3	7.4	4.5	3.0

the first, because the converse is the case. The explanation is probably to be found in the fact that more of the parents had got wise to the possibility of demanding the children back before the time stipulated by law.

If the individual cases are examined separately, some children are found to have had a comparatively short period under care: 10 boys in both periods together had been detained for less than a year. In other cases, the period was remarkably long, and 48 subjects, i.e. more than 20 per cent have been in the charge of the Board for at least 10 years.

The mean period of treatment for the girls in this group is somewhat lower than that for the boys during both periods, particularly the last, when it was a whole year shorter. Nor have quite as many girls as boys been in charge of the Board over considerable length of time. Only 14 per cent have been detained for more than 10 years, while the corresponding figure among the boys was 20 per cent.

For the delinquency cases proper, the period of treatment is found to be longer for the milder cases (Group II) than for

the more serious ones (Group III). The difference stands out plainly during both periods, and to an equal extent for boys and girls. The most natural explanation is that home conditions, contributing as they do to the detention of cases in Group II, must also affect the length of the period of treatment, which must be longer, in the same way as in Group I, where the homes are of the same character as in the second group.

Most interest, however, attaches to the period under care for the more serious cases of delinquency, namely Group III. As appears, the period of treatment for boys in most cases during the earlier period was 3 years, and averaged 2.6 years during the later period. Nearly 10 per cent (37 boys) during the former period, and rather over 10 per cent (29 boys) during the latter have been detained for more than a year, while nearly 15 per cent (54 boys) during the former and rather more than 8 per cent (18 boys) during the latter period had been detained for more than 5 years. Of the former cases, 13 had been in the charge of the Board for more than 7 up to almost 10 years; during the later period however, only 3 had been detained for the same space of time.

A closer survey of the 13 former cases with a longer period under care shows that all were taken in charge when quite young, the average age being 8.6 years. Thus, the boys are almost 4 years younger than the other boys in the same group (average age 12.3 years). That is to say, all showed delinquency at an early age.

As regards the type of delinquency, 3 are crimes against property only, but most of them are combinations of different kinds — in 3 cases of 3 types of delinquency. If a combination of several different types of delinquency is to be taken to indicate more serious delinquency, then these boys had from the very beginning, and despite their strikingly tender years, laid themselves open to lengthy detention.

It is, however, conceivable that the home conditions were such that the boys were kept longer in the charge of the Board. 5 boys came from really bad homes; 3 were from good homes, and nothing was known of the homes of 5. However, 7 of these 13 boys were illegitimate and hardly had a proper home to return to, which may in part explain the long period of treatment.

Of these 13 boys, 7 had at least moderate intellectual powers, 2 were poorly endowed, and there was no information on this head in 4 cases. It is plain, however, that extreme backwardness cannot have been responsible for the long detention in all cases.

The boys from the later period, who had been in a detention home for 7.2 and 9.8 years respectively, had also been taken in charge at an early age (11 years) for repeated thefts. Both were legitimate and with good intellects, but both came from very bad homes, which was probably the main reason for the lengthy detention.

The average period of treatment for the delinquent girls (Group III) was longer than that for the boys during the first period (see Table 45) but the opposite was the case during the later period. There is no very perceptible difference between the period of detention of boys and girls respectively, however. As the table shows, the detention seems as a rule to have been shorter during the second period. It is impossible to determine how far this shorter time can be ascribed to the fact that the lengthy cases belonging to more recent years had not completed their treatment and have therefore not been included in the material.

CHAPTER XIII.

PROGNOSIS.

INTRODUCTION.

A prognosis investigation of delinquent children is interesting for various reasons. It is important, not only from a theoretical viewpoint but also from a practical and a social one, to get some idea of the future prospects of children showing symptoms of social maladjustment even at an early age. But it is of at least equal importance, particularly from a comparative viewpoint, to study the development of children coming from especially bad homes. There are practically no prognosis investigations into this latter category. Previous studies deal exclusively with delinquent children or criminal juveniles, and — with the exception of Ahnsjö — consist solely of an account of the later development over a shorter or longer period of the cases observed by the authors. They are hardly *prognosis* investigations in the real sense of the word, this word implying as it does a probability calculation of the future fate of individuals of the same kind under more or less similar conditions. As a rule, neither the risk figures for different years nor the total risk have been computed. It is necessary to use methods of this kind to arrive at results allowing a uniform interpretation. Despite the statistical lacunae in earlier calculations, they do of course give an approximate idea of the situation, but it is an uncertain and a vague one.

It must be borne in mind that, as the present investigation covers as long a period as 40 years, other opinions and conceptions have come to the fore, new laws have arisen and others have been altered, their interpretation and application have undergone changes — in short, social conditions have not remained the same. The figures hereafter submitted are there-

fore means from a long period of time. A knowledge of the fate of delinquent children necessitates resorting to older material. This has the advantage of providing a longer period of observation, but the unavoidable drawback is that the figures may not express the present state of affairs. It might be suggested that a division should be made over different periods as regards prognosis, also: this would give a more differentiated picture. However, the material is not big enough for such a division.

Needless to say, not all the cases included in the primary material could be followed in respect of prognosis. The prognosis investigation contains 2 206 cases, 1 637 boys and 569 girls, making 94.0 per cent of the total material. (Cf. Table 46.)

The largest contingent of omitted cases consists of the children who were still under detention at the time the investigation was concluded on January 1st 1944, or those, who were admittedly discharged but who were under the age of 15 years at the juncture in question. These latter total 86 cases.

A smaller number, i.e. 19 cases. had died either while still in the charge of the Board or within the months immediately following discharge. 7 further cases fall out, who moved abroad immediately after discharge. Furthermore, we omit those children who were transferred direct from detention home or private home to institutions for permanent care; these numbered 23. Of these, 1 boy had been admitted to an institution for the blind, 6 boys and 2 girls to an institution for educatable imbeciles, and 14 — 10 boys and 4 girls — went direct from the detention homes to lunatic asylums.

The immediate cause of the transference to lunatic asylum was for 5 boys and 2 girls insanity and for 2 boys imbecility (cf. Chapter XI). 1 boys and 2 girls were transferred to lunatic asylum on the diagnosis imbecility+psychopathy. Further, 2 boys have been excluded from the prognosis investigation because they were at the first sentence dismissed without punishment (5: 5). The diagnosis was post-encephalitic change of character, respectively imbecility.

Finally, 5 cases have been omitted who were impossible to trace, making 0.2 per cent of the total number. Thus 140 cases, making 6 per cent, fall out of the primary material.

Cases included in the prognosis investigation have been followed to that part of the country where they were living on

Table 46. **The total number of cases divided into cases not used and used for the prognostic investigation.**

Groups of cases	Period	No. of the original cases	Eliminated cases								Covered by prognosis	
			At discharge					Not discharged	Under 15 years	Total not covered by prognosis	No. of cases	Per cent of the original cases
			Dead	To mental hospitals	To other institutions	To foreign countries	»Unknown»					
Boys												
I	03—25	177	3	—	4	2	—	—	—	9	168	94.9
	26—40	69	—	—	1	—	—	6	19	26	43	62.3
II	03—25	105	2	1	—	1	—	—	—	4	101	96.2
	26—40	61	1	2	1	—	—	14	—	18	43	70.5
III	03—25	396	3	1	1	—	1	—	—	6	390	98.5
	26—40	227	—	4	—	—	—	11	1	16	211	93.0
IV	03—25	324	5	—	—	—	2	—	—	7	317	97.8
	26—40	368	—	2	—	2	—	—	—	4	364	98.9
I—IV	03—40	1727	14	10	7	5	3	31	20	90	1637	94.8
Girls												
I	03—25	178	1	—	—	—	1	—	—	2	176	98.9
	26—40	66	1	—	—	—	—	5	24	30	36	54.5
II	03—25	52	1	—	1	1	—	—	—	3	49	94.2
	26—40	23	1	—	—	–	—	3	—	4	19	82.6
III	03—25	87	—	—	—	—	—	—	—	—	87	100.0
	26—40	160	1	4	1	—	—	3	—	9	151	94.4
IV	03—25	30	—	—	—	1	1	—	—	2	28	93.3
	26—40	23	—	—	—	—	—	—	—	—	23	100.0
I—IV	03—40	619	5	4	2	2	2	11	24	50	569	91.9
Total number		2346	19	14	9	7	5	42	44	140	2206	94.0

January 1st 1944, or, if they died or left the country, up to the year when this took place.

In the course of the years, a number of persons have naturally disappeared into the 'Present whereabouts unknown' books in

the respective parishes. These cases total 23, 20 men and 3 women. However, they could be followed up for a number of years, different in the different cases, and have been looked up in the Penal Register, where 3 of the males and 2 of the females had been registered before the time when they were entered as »unknown» in the Clerical Registers.

After these adjustments, the prognosis investigation has been made along different lines: first in respect of how the subject stands as regards criminality and vagrancy, then in respect of ability to support himself, of temperance, and finally of social status and self-management. With the consent of the Crown, data as to criminality have been obtained from the Central Penal Register. This yielded information on persons sentenced to penal servitude or to prison, fined or released on probation for petty larceny, or receiving a sentence which could not be enforced due to mental condition, as also all persons sentenced to workhouse.

Instead of the usual method of calculating, say the number guilty of crimes during a given time in percent of the number of cases observed *either wholly or partially during* the same period, a more exact method has been taken. That is to say, the risk figure for each year of observation has first been determined, and the total risk then computed for all years.

The number of first offenders during one observation year has been correlated to the number of individuals who run the risk during the same year. A person has been considered to be exposed to risk each year from the time he or she enters the prognosis material till the time when, for one reason or the other, he or she falls out. Consequently, the number of years during which the subject has been under observation will vary, and may for some be only 1 year, and for others up to 40 years. The subject enters the material at the juncture he or she is discharged on probation from the detention home or private home or in regard to Group IV at the time of the sentence (in the case of probation or fine) or at the discharge from reformatory or prison, then embarking on the first year of observation. At the same point in the next year, the subject enters the 2nd observation year, and so on. Any criminal or

other asocial actions during the different years of observation are thus chalked up for the respective years, and the risk figure is calculated in relation to the number of persons exposed to risk. Thus, if a subject is discharged on $^{6}/_{3}$ 1910, the first observation year will run from $^{7}/_{3}$ 1910 to $^{6}/_{3}$ 1911, after which the second year begins. If the subject commits a criminal action on, say, $^{15}/_{3}$ 1911, it will have been committed during the second year, after which the person in question is subtracted from the number exposed to the risk of committing a first offence. Once the subject has appeared as the author of a crime of this kind, he disappears from the material and does not appear again until the calculation, as a possible old offender. The risk figures obtained have then been compared with corresponding figures from the total population.

The years during which the subject was in prison or in reformatory, and consequently unable to commit a criminal action, are subtracted from the observation period. This ought by rights to have been done in the cases of persons admitted to institutions, e.g. sanatoriums, for lengthy illness, thereby having no chance to commit crimes. But is has not been possible to obtain data of this kind, so that a certain source of error is unavoidable in this respect.

The earliest age at which the prognosis investigation started was 15 years, thus coinciding with the age of legal responsibility. Some of the cases in the charge of the Board were under 15 years of age when discharged; needless to say, this applies mainly to the cases from Groups I and II (children detained wholly or partly due to bad home conditions). They do not enter the observation material until they have turned 15 years of age, and the number of persons exposed to risk during the *first* observation year after discharge will consequently be lower than during the years immediately following.

When the risk figures for each year of observation have been computed, a calculation is made of the total risk (according to DAHLBERG, 1943). In the present material, this term is taken to imply the risk run during all the observation years of subjection to measures of some kind, provided the individual in question is alive and exposed to risk for the whole time.

The total crime risk obtained by simple addition is too high, unless the risk figures are very low. The total risk must therefore be computed after subtraction of the number of persons who have committed a crime or some other risked action. As this material has very different observation periods, the calculations have been very laborious.[1]

As is known, the risk of committing crimes differs with different ages, and as DAHLBERG (1943) has shown, is greatest in the ages 15—18 years in the population of Sweden for the year 1937. It should therefore be borne in mind that the assessment of the prognosis for those children included in the present Child-Welfare clientele may be different for those coming under the different periods of 1903—1925 and 1926—1940. Before 1926, the upper limit of the Child-Welfare clientele exactly coincided with the lower limit of the age of legal responsibility; thus, in this part of the material during the earlier period, no one can have been sentenced for criminal actions before intervention by the Board. On the other hand, this might very well happen during the later period (1926—40), when the upper limit of the Child-Welfare clientele was raised first to 18 years and then to 21 years, and the age of legal responsibility thus exceeded.

Nor are the criminal juveniles (Group IV) wholly comparable with the Child-Welfare clientele, in so far as the former had received their first sentence at the juncture when they enter the material. Strictly speaking, then, the criminal juveniles are to be regarded as old offenders the first time they commit a crime during some one of the observation years, while the Board cases only become so if they relapse into crime during the years following their first offence.

A study has then been made of the development of the

[1] When calculating the standard error of the total risk, a method has been used which gives approximate values. Starting from the standard error of the observed risk figures (average annual risk over a 5-year-period), a calculation is made of the standard errors of the annual additions which when squared and added together give the square of the standard error of the total risk. As the correct procedure is comparatively laborious, the above method has been used in this work, though it does give rather too large a standard error.

subject in social respects — first, the extent to which he or she has come into conflict with the vagrancy laws. Data on this head have been obtained from the Police Gazette. Here, the years of observation have been calculated as was done above for criminality. It has naturally been taken into account whether the age of the subject justifies his or her exposure to risk, since the age incurring action from society on one head or the other differs, not only as regards the steps taken but also during different periods of time, due to changes in the law (see Chap. II).

As regards inebriety, data has been collected from the Temperance Boards in the places where the subject resides. Since a number of the men had moved around before their final domicile on January 1st 1944, follow-ups have been made at the Control Board (*Kontrollstyrelsen*), where all drunken offences committed from 1932 inclusive are entered.

To get an idea of how the children in this investigation fare as adult with regard to financial situation and the ability to support themselves and their families, the social register in the respective places has been studied to see whether poor relief has been granted, or if there is any other record of failure to support oneself. Finally, the data as to the callings of the subjects has been used to tabulate their social status according to their occupations, which will then be compared with the corresponding state of affairs for their parents, and with the same distribution in the total population.

After this more general orientation as regards methodics and the cases investigated the prognosis of each group in the material will be dealt with separately.

As has been pointed out previously (Cf. Chapter XI and p. 226 in this chapter) obvious cases of mental illness manifesting themselves during the time of detention have been removed from the material on which estimates of prognosis were made. Naturally certain cases of insanity appeared after discharge. The information that could be obtained on this matter is however very incomplete and is mostly derived from the appropriate social register. 17 such cases were registered in all, 11 men and 6 women, distributed among the different groups. The information is however too insufficient to be useful in

estimating the actual risk, and even if it was checked with the various hospitals where the individuals were admitted and from which the diagnoses were obtained, it must be remembered that this investigation covers so long a period that the diagnostic systems in use at the beginning were quite different from those in use now. For this reason, and because the number of cases are so few, any calculation of risk would be of but little interest, specially as there is very little information as to the risk of mental illness among normal populations so that no comparison could be made.

There is one matter, however, which may be of interest in this connection. It is to what extent the affected offenders were dismissed without punishment (5: 5) or were considered to have diminished responsibility (5: 6). According to Swedish penal law in force at the time imbeciles, and those who, by reason of illness or the infirmity of age, are deprived of the use of reason, are not punished. A reduced penalty may be imposed on those who, at the time of committing the offence, were lacking the full use of reason owing to bodily or mental illness.

The number of cases in the material used for the estimation of prognosis to which the first of these paragraphs was applied was 25 altogether, 22 men and 3 women. As the numbers are so small, no calculations of risk have been made here either, but we have contented ourselves with giving the number of cases and the causes.

The diagnosis was imbecility or psychopathia either separately or combined in 16 cases. In 2 cases it was insanity caused by a cerebral lesion. There was also one diagnosis of infantile asthenia and one of schizophrenia. In the rest of the cases the diagnosis is not known.

3 of the women in this material who committed crimes were dismissed without punishment, one under the diagnosis of schizophrenia and the others under that of imbecility.

10 cases were described as having diminished responsibility. In 4 cases the diagnosis was given as psychopathia and in one case imbecility. In the rest the diagnosis is not known.

CONDUCT DURING THE TIME AFTER BEING TAKEN IN CHARGE AND BEFORE THE AGE OF 15 YEARS FOR CHILDREN COMING FROM BAD HOMES (GROUP I).

As appeared from the previous chapter, all the children in this group were taken in charge for prophylactic ends, due to poor home conditions,

As already stated, there is reason to consider this group potentially delinquent in view of the particularly bad home environment, and, moreover, in view of the less favourable heredity with which, according to the foregoing investigation, these children are undoubtedly encumbered.

When taken in charge, however, they showed no signs of maladjustment, whether this was because they were so young or because, despite appearances, their disposition was not such. The children in this group are thus different from the other groups in this respect; the prognosis investigation proper, which begins at the discharge of the children and at earliest at the age of 15 years when the age of legal responsibility is reached, may therefore justifiably be preceded by an investigation into the development of these children during the years immediately following the action by the Board. In a way, these data, too, are of prognostic character.

Table 47. **Frequency of delinquency in children from bad homes (group I) after taken in charge but before 15 years of age.**

Period	Group I No. of cases = N	Cases of delinquency		Cases transferred to detention homes	
		No.	in per cent of N	No.	in per cent of N
		Boys			
1903—25	174	59	33.9 ± 3.6	9	5.2 ± 1.7
1926—40	60	17	25.8 ± 5.4	4	6.1 ± 2.9
1903—40	240	76	31.7 ± 3.0	13	5.4 ± 1.5
		Girls			
1903—25	177	32	18.1 ± 2.9	14	7.9 ± 2.0
1925—40	64	12	18.8 ± 4.9	7	10.9 ± 3.9
1903—40	241	44	18.3 ± 2.5	21	8.7 ± 1.8

This investigation consequently includes part of those children who have been subtracted from the final prognosis because they were under age, or still in their foster-homes on $^1/_1$ 1944. Thus, only 6 boys have now been deducted from the total material, of whom 4 had been admitted to an institution for permanent treatment before the age of 7 years, and 2 were under 7 years old on January 1st 1944.

3 girls drop out for the same reason. Thus, this part of the investigation comprises 240 boys and 241 girls, making 97.5 per cent and 98.7 per cent respectively of the entire number in the group.

Needless to say, the length of the observation period will vary, since a number drop out between 7 and 15 years for different reasons. This does not play much part in these cases, however, since the investigation aims solely at seeing whether any of these children exhibited delinquency, and if this was serious enough to occasion transference to a detention home, all these children having been placed direct in private homes, mostly in the country.

We then find that, in round numbers, every 3rd boy (31.7 per cent) and every 5th girl (18.3 per cent) displayed delinquency symptoms, if the children from both periods are put together (Table 47). It is difficult to judge how serious these delinquency tendencies were. It must be borne in mind that it may be in the interests of the foster-parents to complain. Nevertheless, an objective indication that the offences were not unduly slight is given by the fact that 13 of the boys (5.4 ± 1.5 per cent) and 21 of the girls (8.7 ± 1.8 per cent) were transferred to detention homes. Compared with the approximate frequency figure for the average population elicited from this investigation, i.e. 2 per cent for boys and 1 per cent for girls, we find the former figures nearly twice as much for the boys and 8 times as much for the girls. The difference (3.3 ± 2.3) between boys and girls is not statistically significant, and may be due to chance. Fewer girls than boys have shown symptoms of delinquency, but it seems as if the frequency of graver delinquency, necessitating treatment in a detention home, is commoner among girls than among boys. However, different judgments of maladjustment among the girls probably explain

the stricter action taken in their case. Sexual offences among girls are always frowned upon and regarded as a more serious dereliction than, say, running away or theft among boys. A closer inspection of the cases in question may throw light on the situation in these cases.

A survey of the offences immediately prompting the transference of these children to detention homes shows that, in the case of the boys, practically all concerned combinations of several types of delinquency. General delinquency predominates, though it is never the sole reason, appearing together with pilfering, wanton damage or cruelty to animals. 3 of the boys have attempted arson.

For the girls, general delinquency is similarly a prevailing feature, with symptoms such as mendacity and untrustworthiness, malevolence and insolence. Petty larceny is also a frequent feature. In addition to symptoms of this kind, 9 girls showed signs of maladjustment in sexual respects. The note against 4 of these is 'strong sexual interests', against 2 'running round the streets', against 1 'shaky morals', and against 2 that they had had intimate relations with the farm-hands.

If we subtract these 9 girls, who had been rather severely condemned for their offences, we are left with only 12, who had been transferred to detention homes for the same kind of offences as the boys. It is therefore clear that more serious delinquency of other kinds appeared about as often among the boys as among the girls.

The average age at which these children were transferred to detention homes was 13 years, about the same for boys as for girls.

The data are indefinte as to the interval between detention and the first appearance of the delinquency symptoms. On an average, 4—5 years elapsed between the two, about the same for boys as for girls. This interval naturally depends on the age at which the subjects were taken in charge, since real delinquency symptoms can only appear at higher ages, so that the interval must be longer, the earlier the child was detained.

Another more interesting matter is whether these children showing delinquency — and particularly those transferred to detention homes — were taken in charge later than those not showing signs of maladjustment. As regards the boys, those showing delinquency were on an average taken in charge at the age of 7 years, and those subsequently transferred to detention homes even earlier, about 6 years, while those who

behaved well during their period of detention had been taken in charge at the age of 8 years. The same applies to the girls. Those who showed delinquency had all been taken in charge at the average age of 7.7 years, and the other group at 8.3 years. However, the difference is not significant either for boys or girls, and is especially small for the girls. Thus, no connection can be shown between the age when taken in charge and later manifestations of delinquency (if any). We might point out, however, that this only applies to the state of affairs in this group with the variation obtaining here in respect of the juncture when the subjects were taken in charge.

It can be said here and now that of the boys taken in charge during the first period and showing delinquency symptoms 22 of 59 cases (37.3 ± 6.3 per cent) were convicted of crimes later in life, while this only applies to 12 of the 115 boys with apparently good conduct during detention (10.4 ± 2.8 per cent). The difference is 26.9 ± 6.9 per cent, and is significant.

The same is true of the girls from the same period. Of the 145 not showing signs of delinquency, 6 were later convicted (4.1 ± 0.65 per cent), whereas 2 of the 32 with signs of delinquency were subsequently sentenced for criminal actions (6.3 ± 4.3 per cent). The difference of 2.2 ± 4.4 per cent is not significant, however. These figures are given only for the sake of completeness.

Having now given a general survey of the conduct of the children during the years after they were taken in charge and before they reached the age of 15 years — the age of the 'criminal majority' — we will examine the later development of these children in different respects.

PROGNOSIS OF CHILDREN FROM BAD HOMES. (GROUP I)

As shown in the introduction, not all cases from the primary material could be used for the final prognosis. Group I loses 35 boys and 32 girls, for different reasons (see Table 46); there thus remain 211 men and 212 women, taken in charge when children for prophylactic ends on the score of risk of delin-

quency. Of the cases remaining for the prognosis, 168 men and 176 women are from the first period, and 43 men and 36 women from the second.

1. Prognosis as regards criminality.

We shall first investigate how these persons stood as regards criminality. If we only take into account the number subsequently convicted of crimes of the previously mentioned kind — i.e. Central Penal Register crimes — we get for the men a frequency figure of 20.1 per cent (34 cases) from the earlier period, and 13 per cent (6 cases) from the later one. This difference, which at first sight suggests greater criminality for the men of the earlier period, can without doubt be ascribed to the different length of the period of observation, which must naturally be shorter for those coming from the later period.

To eliminate this source of error, and to obtain a correct picture of the situation at all, it is necessary to use risk calculations for different years and different ages. Such have consequently been made, and used to compute the total risk, up to a certain age in order to make possible a comparison with the normal population, for which there are risk figures and computed total risk for the year 1937. DAHLBERG (1943) having used material from this year to calculate risk figures.

The first question to arise then is: How far is this year representative of the situation in Sweden?

The rate of criminality varies and is especially high during crises and war years. The »normal» figure for the population therefore depends on which year the figures are taken from. Further, the risk of criminality is distributed in different ways on the different age groups. Earlier the risk was highest for the age group 18—21 years. After 1935, the risk was highest for the age group 15—18 years. Under such circumstances a comparison with DAHLBERG's risk figures — the only risk figures existing for the Swedish population — must be made with a certain caution, especially for the younger age groups.

Table 48 and Fig. 10 show anyhow that 1937 was a fairly average year in the respect here discussed. The risk of criminality in this year kept at the mean level of the 20-year period

Table 48. **Absolute number and relative number in per thousand of the average population of persons who were convicted by lower court during the years 1915—1943 and entered in the Penal Register.**

Year	During the year entered in the Penal Register		In per thousand of average population in age of criminal majority	
	Men	Women	Men	Women
1915	4610	430	2.45	0.21
1916	5240	436	2.75	0.21
1917	7318	752	3.78	0.36
1918	12612	1148	6.45	0.54
1919	6247	752	3.16	0.35
1920	4267	497	2.13	0.23
1921	3724	397	1.83	0.18
1922	3226	315	1.57	0.14
1923	3284	297	1.58	0.13
1924	3073	312	1.47	0.14
1925	3700	349	1.74	0.15
1926	3442	342	1.61	0.15
1927	3667	374	1.69	0.16
1928	3696	416	1.69	0.18
1929	4094	398	1.85	0.17
1930	4193	448	1.88	0.19
1931	4400	448	1.95	0.19
1932	4673	482	2.04	0.20
1933	4772	464	2.06	0.19
1934	4798	634	2.05	0.26
1935	5066	477	2.14	0.19
1936	5216	486	2.17	0.20
1937	5376	486	2.22	0.19
1938	5724	529	2.34	0.21
1939	6596	495	2.68	0.19
1940	6495	644	2.61	0.25
1941	8100	1087	3.23	0.42
1942	10467	1522	4.15	0.59
1943	10069	1308	3.96	0.50

1921—1940. Nor was there any special economical crisis at the time. The general risk figures from this year are therefore tolerably comparable with the risk figures for this material, particularly as there are special figures for Malmö.

As already mentioned, the material was used in its entirety

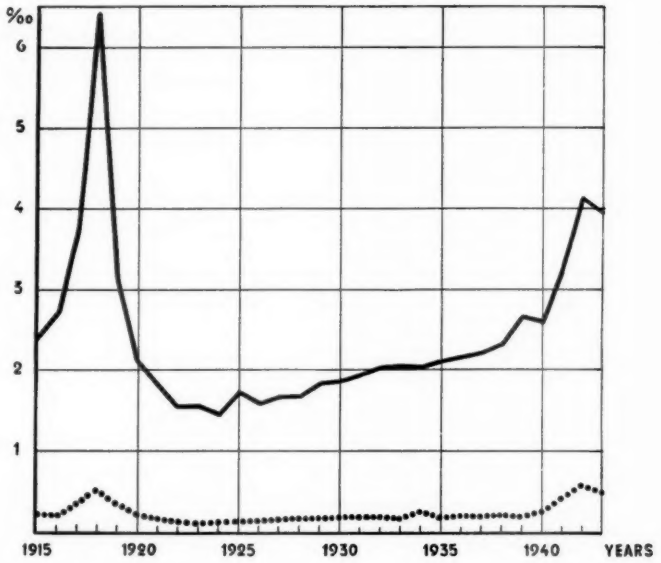


Fig. 10. The annual frequency of persons (in per thousand of the average population 15 years of age and over) who are convicted of serious crimes (recorded in the Penal Register). Whole line = men, dotted line = women.

in each group at the prognosis investigation, and no division into periods has been made.

If we begin by comparing the annual risk figures for the men in this group with DAHLBERG's figures for Malmö, we find such good agreement for youths aged 15—18 years that these persons can almost be said to have shown no greater criminality than people in general.

However, the annual risk figure rises in the following 3-year group, reaching its highest value (2.7 ± 1.9 per cent) between the ages of 18—21 years, where it is now considerably above the normal figure (0.3 per cent for 18—21-year-olds). See Table 49. The following age groups, as also the normal population, yield slowly falling risk figures. The important thing is, however, that with the exception of the above-mentioned ages,

Table 49. **Average annual risk in per thousand and calculated total risk in per cent of the average population of being convicted of crimes recorded in the Penal Register for persons in different age groups, on the basis of the conditions obtaining in 1937.** (After DAHLBERG, 1943.)

Age, years	Men				Women in towns	
	Average annual risk per thousand		Calculated total risk per cent		Average annual risk per thousand	Calculated total risk per cent
	Malmö	Towns (Sweden)	Malmö	Towns (Sweden)		
15—18.....	13.41	9.20	3.97	2.74	0.65	0.20
18—21.....	3.42	5.84	4.95	4.43	0.71	0.41
21—25.....	2.82	3.86	6.02	5.90	0.66	0.67
25—30.....	2.51	2.88	7.19	7.24	0.32	0.82
30—35.....	1.10	2.23	7.70	8.27	0.21	0.93
35—40.....	1.62	1.80	8.44	9.10	0.18	1.02
40—45.....	0.83	1.28	8.82	9.68	0.06	1.05
45—50.....	1.10	1.14	9.32	10.19	0.13	1.11

the annual risk figures are appreciably higher in this group than for the normal population. On the other hand, the particularly high figure for 35—40-year-olds may well be due to chance and to the large standard error.

As appeared from DAHLBERG's investigation (1943; see Table 49), the annual risk figure for the year 1937 is highest in the total population between the ages of 15 and 18 years. The main concentration of cases in this group is found among 18—21-year-olds. This is probably because the risk is increased for younger ages towards present time. A great number of the cases belong to older periods when the normal risk for the young of being criminal was smaller. This seems to be the main cause of the difference.

If we calculate the total risk on the annual risk figures, and compare the result with DAHLBERG's above-mentioned calculations of the total risk threatening the normal population in Malmö, we find that the figures for 18-year-old men still agree well, but that they then increase considerably, and are 3 times the normal value for men turning 45 years. The difference as regards the standard error is significant. (Table 50.)

Table 50. **Average annual risk and calculated total risk in per cent for men of committing a crime, being reported to the Temperance Board because of inebriety, being warned or sentenced for vagrancy or of having to apply for poor relief. Group I.**

Age	Criminality	Age	Inebriety	Vagrancy	Poor relief
		Average annual risk			
15—18.....	1.3 ± 0.8	15—20.....	0.2 ± 0.3	0.3 ± 0.4	1.1 ± 0.7
18—21.....	2.7 ± 1.2	20—25.....	1.0 ± 0.8	1.0 ± 0.8	1.9 ± 1.1
21—25.....	1.4 ± 0.9	25—30.....	0.3 ± 0.5	0.1 ± 0.3	1.3 ± 1.0
25—30.....	0.5 ± 0.6	30—35.....	0.9 ± 1.0	0.2 ± 0.4	2.4 ± 1.5
30—35.....	0.2 ± 0.4	35—40.....	—	—	2.5 ± 2.1
35—40.....	1.3 ± 1.5	40—45.....	·	—	1.6 ± 2.5
40—45.....	—				
		Calculated total risk			
18.....	3.9 ± 1.4	20.....	1.0 ± 0.8	1.5 ± 0.8	5.5 ± 1.6
21.....	11.5 ± 2.4	25.....	5.9 ± 2.0	6.4 ± 1.9	14.2 ± 2.8
25.....	16.3 ± 2.9	30.....	7.4 ± 2.2	6.9 ± 1.9	19.7 ± 3.3
30.....	18.3 ± 3.1	35.....	11.4 ± 3.1	7.9 ± 2.1	28.8 ± 4.3
35.....	19.3 ± 3.2	40.....	11.4 ± 3.8	7.9 ± 2.5	37.2 ± 5.5
40.....	24.3 ± 4.2	45.....	·	7.9 ± 3.7	42.1 ± 6.5
45.....	24.3 ± 5.0				

For the 15—18-year-old *females* in this group (Table 51), however, the annual risk figure is nearly 10 times the normal figure (i.e. only 0.06 per cent) for women from towns. Consequently, the total risk is also high: for women turning 40 the total risk is about 5 times that of women in the normal material. The material is small, however, so that the difference is not fully significant.

In connection with the above figures, it may first of all be stated that the increase vis-à-vis the normal population seems to be greater for the women than for the men. In spite of this, the prognosis shows that appreciably fewer women than men turn criminal. To judge from the figures, boys running the risk of delinquency are at least 3 times as likely to become criminals than girls in the same situation (up to the age of 30).

The question now is whether any of these individuals were *re-convicted*, and whether this happened more than once. We

Table 51. **Average annual risk and calculated total risk in per cent for women of committing a crime, being warned or sentenced for vagrancy or of having to apply for poor relief. Group I.**

Age	Criminality	Age	Vagrancy	Poor relief
		Average annual risk		
15—18	0.5 ± 0.5	15—20	0.2 ± 0.3	0.2 ± 0.3
18—21	0.2 ± 0.3	20—25	0.3 ± 0.4	0.7 ± 0.6
21—25	0.4 ± 0.5	25—30	—	0.5 ± 0.6
25—30	0.3 ± 0.4	30—35	—	0.9 ± 0.9
30—35	—	35—40	—	0.3 ± 0.6
35—40	—			
		Calculated total risk		
18	1.5 ± 0.8	20	1.0 ± 0.7	1.0 ± 0.7
21	2.1 ± 1.0	25	2.0 ± 1.1	4.5 ± 1.5
25	3.7 ± 1.4	30	2.0 ± 1.3	7.0 ± 2.0
30	5.2 ± 1.7	35	2.0 ± 1.5	11.0 ± 2.7
35	5.2 ± 1.9	40	2.0 ± 1.9	12.5 ± 3.0
40	5.2 ± 2.1			

find that 12 of the 40 men and 2 of the 9 women convicted of a first crime subsequently sinned a second time, and that 8 of the men and 1 of the women were further sentenced a third time.

However, this relapse applies solely to persons who were taken in charge during the earlier period. None of those from the later period has been convicted of more than one crime — probably because they have not had time to become as old as the men and women of the first period.

The risk of relapse seems to be greatest in the first year after the initial crime. Of the 8 men offending again within 5 years, 5 fall within the first year. This tallies with results from previous investigations of larger materials (DAHLBERG and LINDBERG 1944), namely, that the risk is greatest the first year after discharge. (Table 52.)

The total risk of relapse is also fairly high, and the table shows that nearly one third of the men offended again within 10 years from their first crime.

Table 52. **Average annual risk and calculated total risk in per cent of relapsing into crime during different periods after committing a first crime entered into the Penal Register.**

a. Men.

Years	Group I	Group II	Group III	Group IV	
				Crime No. 2	Crime No. 3
Average annual risk					
1— 5	5.3 ± 4.1	10.8 ± 6.1	14.6 ± 3.1	6.4 ± 1.0	14.8 ± 3.1
6—10	1.9 ± 3.0	4.3 ± 4.7	3.2 ± 2.1	1.3 ± 0.6	3.2 ± 2.2
11—15	·	·	1.9 ± 1.9	1.0 ± 0.6	2.0 ± 2.2
16—20	·	·	0.5 ± 1.2	0.3 ± 0.4	1.5 ± 2.4
21—25	·	·	—	0.8 ± 0.7	—
Calculated total risk					
5	23.9 ± 9.1	43.5 ± 13.6	54.6 ± 7.0	28.1 ± 2.3	55.1 ± 7.0
10	30.8 ± 10.5	54.5 ± 14.8	61.5 ± 7.4	32.6 ± 2.5	61.8 ± 7.4
15	·	·	65.0 ± 7.5	36.0 ± 2.7	65.4 ± 7.6
20	·	·	66.0 ± 7.6	37.0 ± 2.7	67.9 ± 7.8
25	·	·	66.0 ± 7.7	39.5 ± 2.9	67.9 ± 8.0

b. Women.

Average annual risk			*Calculated total risk*		
Years	Group III	Group IV (Crime No.2)	Years	Group III	Group IV (Crime No.2)
1— 5	1.0 ± 2.2	4.7 ± 3.2	5	5.0 ± 4.9	21.5 ± 7.2
6—10	—	0.6 ± 1.4	10	5.0 ± 10.0	24.0 ± 7.6

The figures are based on a very small material and carry large standard errors, so that no very great value can be attached to them. A comparison with the relapse figures submitted by DAHLBERG and LINDBERG (1944) yields no significant differences, although it seems as though the risk of relapse in this material were rather less than for criminals in general. But a larger material is required for definitive conclusions.

It can thus be established that children taken in charge on account of bad home conditions run a considerably greater risk of subsequently committing a Penal Register crime than do children in general.

It may, however, be interesting to see what kind of crimes these individuals have committed. As always, most of them are crimes against property: in most cases theft, in some cases theft combined with housebreaking, and in some in the form of petty larceny, making 26 cases in all. There are 4 cases of fraud or forgery. 4 have been court-martialled and imprisoned. Sexual offences are found in 2 cases, and 4 cases have been sentenced for different crimes and misdemeanours, such as illicit peddling, violation of domicile, and unlawful 'borrowing' (2 cases). Sentence was remitted in 2 cases, one on account of psychosis (schizophrenia), the other on account of imbecility.

Only one boy in this group has been admitted to a reformatory. His career was briefly as follows:

No. $^{11}/_{8}$. Hans, illegitimate; separated from his home at the age of 3 years. Parents not married but living together. Father sentenced several times for stealing; was drunken, rough, and unwilling to work. Mother dull-witted and unreliable; used to go round begging. The home was singularly wretched and uncared-for; highly unsatisfactory. Attempts by the Poor Relief Board to secure them better quarters had no result, since the family was too indolent to move. The children were inadequately fed and clothed. The boy, who was placed in a good farmer's home, developed normally for the first 7 years, but began about the age of 10 to show signs of delinquency in the form of insolence and wanton mischief, and bad conduct generally. He was transferred to a detention home at the age of 13 years, from which he ran away 3 years later, and wandered about for some years as a vagabond. He was then admitted first to a more stringent detention home and then direct to a reformatory, where he spent 2 years. No subsequent criminal behaviour recorded, but he died from tuberculosis 2 $^{1}/_{2}$ years after discharge.

Most of the 12 relapsing into crime were guilty of theft; in addition 2 committed fraud and 2 assault and battery. 8 of these, as mentioned above, were subsequently guilty of further crimes and can be accounted habitual offenders.

Of the 9 women, 4 were sentenced for petty larceny and 3 for plain stealing, while 1 was guilty of fraud and 1 of arson (the girl mentioned on p. 197). One of these 9 women was again detained for educative purposes, but this time in a reformatory. Her history was briefly as follows:

No. $^{10}/_{21}$. Brita S., 2nd of 4 sisters, who were all taken in charge at the same time, as the mother had died and the father was a drunkard. The home

was very poor; unreasonable father, living with a woman who did not bother about the children. Brita was 8 years old when taken in charge, and only a year later began to lie and steal, and be very unreliable. After 2 years in a foster-home she was transferred to a detention home, and after 6 years there she was sent straight to a reformatory. The total period of detention was 9 years. After discharge she led a particularly slovenly and disorderly life. Is now married and living in a large town. Has been sentenced 4 times for petty larceny, theft with housebreaking, forgery, etc. Has been in the workhouse for vagrancy. In addition she has been addicted to alcohol and been prosecuted for drunkenness. She must therefore be regarded as socially impossible. We may add that one of the sisters taken in charge at the same time has also been convicted of crimes according to the Central Penal Register.

No abnormal traits of temperament were entered against any of the girls, and 6 have been considered of normal intellect. Only 1 was feeble-minded, and there was no data as to the mental powers of the remainder.

2. Prognosis as regards temperance.

An important side in a subject is his attitude to alcohol. A person who perpetually takes too much and is seldom sober is in Sweden taken charge of by official institutions, the Temperance Boards. These were set up in 1916. The habits of persons reported to the Temperance Board are gone into closer. If the charge proves justified, measures are taken: in mild cases only a caution, in worse cases supervision or detention. Investigations by DAHLBERG (1942) have elicited the average risk for the town population in this respect. We would point out that, in the country, the Temperance Boards of earlier days stepped in more by way of exception.

In Sweden, if a person has appeared visibly intoxicated in a public place, he is taken in charge by the police and then sentenced for drunkenness. All such offences have been registered since 1932 with the Control Board (*Kontrollstyrelsen*). There are normal figures for a Swedish population on such matters, also. (See DAHLBERG, 1944.)

Thus, the two measures on which we have collected information are intervention by the Temperance Board and sentence for drunkenness. The men included in the material have thus been

investigated on both these heads, while the women have been left out of account, since offences of this kind by women are extremely few and an investigation on so small a material would not be worth while.

As Table 50 shows, the men in this group seem to be exposed to a relatively small risk of incurring measures from the Temperance Board. The most usual age seems to be between 20 and 25 years, and the total risk for men reaching the age of 35 years is 11.4 ± 3.8 per cent. When these figures are compared with DAHLBERG's figures for the total population of Malmö (1942), we see that they are considerably higher than normal.

As seen, the annual risk in the ages between 15 and 20 years is about the same for the men in this group as for those in the total population 1934—38, and is highest for 18—21-year-olds — which can probably be ascribed to chance — whereas the highest risk figure among the total population is not found until the ages between 40 and 50 years.

The total risk for Malmö men turned 40 years was about 4.3 per cent for the years 1929—1933, and 6.1 per cent for 1934—1938. Compared with these, then, our corresponding figure is about double. (Cf. Table 53.)

Naturally, the frequency of offences of drunkenness is only a rough gauge of the sobriety of a group of individuals. Now, we have no data as to the frequency of drunkenness in earlier days. In addition, it was at that time comparatively usual for sentences of this kind to be remitted. It is only from 1932 inclusive that we have had a registration of offences of drunkenness covering the entire country. It has admittedly been possible to obtain data as to a number of such offences even for the time before this juncture, but not until after 1932 has it been possible to make comparisons with the normal population. We shall therefore refrain from data as to conditions in older times, and merely submit data for cases born 1917 and later.

If, using the risk figures as usual, we compute the total risk, we get the figure 2.5 ± 2.5 per cent for the 20-year limit, and a total risk of 11.4 ± 7.0 per cent for the 25-year limit. Thus, the latter figure in particular is somewhat higher than the figure for the average population: which in 1938 was 7.3 and in 1942

Table 53. **Percentual risk for men in Malmö during the years 1929—33 and 1934—38 of being reported to the Temperance Board.** (After DAHLBERG, 1942.)

Average annual risk			Calculated total risk		
Age	1929—33	1934—38	Age	1929—33	1934—38
15—20.....	0.03	0.17	20	0.13	0.85
20—25.....	0.11	0.21	25.....	0.69	1.91
25—30.....	0.16	0.20	30.....	1.49	2.89
30—35.....	0.24	0.27	35.....	2.68	4.19
35—40.....	0.34	0.40	40.....	4.34	6.12
40—45.....	0.39	0.39	45.....	6.21	7.96

10.9 per cent. However, our figure is based on so small a material that the standard error will be very large; the difference is therefore not significant.

3. Prognosis as regards vagrancy.

Vagrancy has always been accounted one of the symptoms of maladjustment to society and of instability generally. It is therefore important to study how the individuals in this group stand in this respect, when adult. We should first mention that, as the material is small, the calculation does not differentiate between the two separate measures of caution for vagrancy or the work-house.

Thus, 14 of the 211 men in this group have incurred measures of this kind.

The annual risk for the men in this group is highest for 18—21-year-olds and then remains at about the same level for men between 21 and 25 years of age, after which it falls fairly rapidly. Sentence for vagrancy has been most usual in this clientele at the ages 18—25 years. In the very youngest ages, 15—18 years, action has been less common; that is to say, prosecution or sentence occurs more rarely. This is no doubt partly because the authorities are disinclined to take such measures against persons in these ages, and because bad conduct of the kind that is called 'vagrancy' for adults is probably called 'delinquency' in these ages, and leads directly to intervention by the Child-Welfare Board. (Cf. Table 50.)

The total risk of action against vagrancy threatening men who have reached the age of 40 years is 7.9 ± 3.7 per cent. These figures can be compared with figures obtained in an as yet unpublished investigation by DAHLBERG from the State Institute of Human Genetics. These latter figures, which apply to the total population — married and unmarried — of all the towns in Sweden, show that for the age group 21—25 years the annual risk of a first caution for vagrancy was 0.05 per cent in 1932, but only 0.01 per cent in 1939. The risk figures for the men in our corresponding age group are thus more than 10 times as high. The total risk for men of 50 years in our group is also extremely high compared with the normal figures, which amounted to 0.8 per cent in 1932, and to only 0.3 per cent in 1939.

Only a few of the women in this group incurred action of this kind, namely 4 out of 217 — that is to say, too small a number to make a calculation of the annual risk worth while; we can only say that it would be very low. (Cf. Table 51.)

4. Prognosis as regards self-support.

The next important question is how far the subjects have proved able to support themselves, or how far they have had to appeal for public assistance. Even if data on these heads have been obtained from official quarters, i.e. from the Social Register and, for earlier times, from the records of the Poor Relief Board in the places where the individuals lived, it is not easy to assess them. An investigation must take into account not only the reason why relief was granted but also the amount granted, as also whether it continued unbroken over a longer period or was only temporary. It goes without saying that isolated, occasional need of assistance in sickness or unemployment does not constitute the same indication of asociality as long-continued relief due to laziness. We can further assume that relief was paid out according to quite different norms in different places, differing in town and country. It may also be said that such norms were doubtless very much more exacting formerly than at present. It should there-

fore be emphasized that the data are difficult to evaluate, all the more so as there are no figures for the normal population with which to make comparisons. No grading of the social aid has been possible; all that has been done has been to tabulate whether or not such help was granted. This help may consequently vary from full support to more temporary assistance, and the data must be regarded as maxima. In any case, however, we can take it that, when help was granted, the subjects in question were at least balancing on the limit of an existence minimum.

Of the entire number investigated, more than every 4th man (57 men) and not quite every 10th woman (19) had received relief in some form; thus, considerably more men than women.

This is reflected more correctly in the annual risk figures (cf. Table 50). About 1 per cent of the males in the younger age groups between 15 and 30 years were in receipt of annual relief, and double this number at the ages between 30 and 40 years.

The total risk for men reaching the age of 45 years is 42.1 $\pm$6.5 per cent, showing that almost half of the men in this group receive relief if they live to be 45 years of age.

As already pointed out, the risk for women in this group is considerably lower, and the total risk for women is $^1/_3$ that for men of the same age. This is no doubt due, at any rate in part, to the fact that most of the women marry, so that if they fall ill or are otherwise incapacitated from working, it will be the husband who has to look ofter them, society only intervening if he is absent or neglectful. (Cf. Table 51.)

The fact that relief has been granted to subjects as young as 15—20 years suggests it was prompted by disabling diseases of some kind. It is therefore of interest to study the special reasons for defective powers of self-support in the different cases.

Of the 57 men, only 20 received occasional aid, while 37 were granted relief in a more permanent form — in at least 2 times in 2 years. Relief was extended to 6 of these on the grounds of disabling diseases such as the aftermaths of infantile paralysis, spondylitis, epilepsy in 2 cases, chronic polyarthritis and spastic paralysis.

Receipt of relief in the case of the women has not the same implications as in that of the men. For the married woman, it actually only means that the husband is unable to support the family. It may therefore be interesting to see whether the relief was granted to married or to unmarried women, and we find that 18 were married, and that all have been in receipt of relief for several years. Only one was unmarried, and had been granted relief during sickness.

On comparison, relief is found to have been granted to about 5 times as many of the men (26.6 per cent) and 1 $^1/_2$ times as many of the women (8.7 per cent) in this material as to the entire population of Malmö (men and women together) during the years 1903—1923 and 1928—1938; these latter figures were 5.1 per cent and 5.8 per cent respectively.

If we go on to compare these individuals with their parents, we find that, during the period 1926—1940, the fathers were in receipt of relief as often as their sons (21.7 per cent as against 22.2 per cent).

The different materials above are not wholly comparable. There are undoubtedly differences in age, as also in other respects. The comparison can therefore only hope to give a very approximate view.

All the same, it is clear that a relatively large number of the males who, as children, were taken in charge due to bad homes, proved when adult to be less capable of supporting themselves and more in need of social assistance than the town population in general.

5. Social Position.

It is also interesting to study what social position these children achieved as adults, and to compare it both with that of the parents and that of the normal population. Here, regard must of course be paid to age. The limit has been set at 25 years, which for men in the social classes covered by this material can be taken to be the age when the callings should have become more or less stable. Of the men whose callings were stated, 29 were under and 161 over 25 years of age on January

Table 54. **Comparison between the social position of parents and sons. Percentual distribution.**

Social class	Group I 121 families		Group II 88 families		Group III 377 families		Group IV 446 families	
	Parents	Sons	Parents	Sons	Parents	Sons	Parents	Sons
I	—	—	1.1 ± 1.1	—	0.5 ± 0.4	—	—	—
II	5.8 ± 2.1	15.7 ± 3.3	4.6 ± 2.2	10.2 ± 3.2	13.3 ± 1.7	13.3 ± 1.7	17.0 ± 1.8	16.8 ± 1.8
III ...	87.6 ± 3.0	80.2 ± 3.6	87.5 ± 3.5	81.8 ± 4.1	82.8 ± 1.9	78.5 ± 2.1	81.6 ± 1.8	78.7 ± 1.9
IV ...	6.6 ± 2.3	4.1 ± 1.8	6.8 ± 2.7	8.0 ± 2.9	3.4 ± 0.9	8.2 ± 1.4	1.3 ± 0.5	4.5 ± 1.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

1st, 1944, when the investigation was concluded; corresponding figures for the women were 30 and 157. In order to make a comparison with the social status of the parents, however, only those cases are accounted for in Table 54 in regard to whom it has been possible to estimate, according to the information obtained, the social status of both the parents and of the adult children.

The distribution into social classes according to different occupations has been done on the same principles as were followed when classifying the population on their stated callings in the election statistics of 1940, and which have also been used by the author before.

When the men in this group were divided up, about 80 per cent of those over 25 years old were found to belong to social class III and 4 per cent to social class IV, while 16 per cent had attained to social class II. If classes III and IV (this latter class being added by the author) are made into one, and a comparison made with the total population, a significant difference is obtained, i.e. 22.2 ± 3.3 per cent, so that more men in this group belong to class III than we should have expected. There is also a significant difference as compared with class II, inasmuch as not quite so many belong to this latter class as comparison with the total population would lead us to expect.

The women over 25 years old in this group have got rather higher up the social scale than the men (cf. Table 55). That is to say, they outnumber the men in class II (22.3 ± 3.8 per

Table 55. **Comparison between the social position of parents and daughters. Percentual distribution.**

Social class	Group I 121 families		Group II 39 families		Group III 126 families		Group IV 31 families	
	Parents	Daughters	Parents	Daughters	Parents	Daughters	Parents	Daughters
I	—	0.8 ± 0.8	—	—	—	—	—	—
II	9.1 ± 2.6	22.3 ± 3.8	2.6 ± 2.5	5.1 ± 3.5	6.3 ± 2.2	15.1 ± 3.2	22.6 ± 7.5	19.4 ± 7.1
III ...	87.6 ± 3.0	74.4 ± 4.0	87.2 ± 5.4	82.1 ± 6.1	88.9 ± 2.8	77.8 ± 3.7	77.4 ± 7.5	80.6 ± 7.1
IV ...	3.3 ± 1.6	2.5 ± 1.4	10.2 ± 4.8	12.8 ± 5.4	4.8 ± 1.9	7.1 ± 2.3	—	—
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

cent), and are fewer than the men in class III (74.4 ± 4.0 per cent). Here, too, there is a significant difference vis-à-vis the total population, which shows that the women, too, have not quite reached the level here, but are somewhat lower socially.

It may be of interest to compare the social position of the children when grown up with that of the parents. As appears from the table, both sons and, in particular, daughters, have bettered themselves socially. It is true that none of the sons belongs to social class I, but then, neither did any of the fathers. Several sons have attained to class II, and the difference as compared with the fathers is probable (9.9 ± 3.9 per cent).

This difference is even plainer for the women. None of the fathers came under class I, but one of the women has reached there, and several of these women belong to class II, while a smaller number are from classes III and IV. The difference is not significant as regards the latter class, however. Now, the social position of the women is very dependent on the position of the man, and is therefore linked up with marriage, so that it does not constitute any real indication of personal capability in the usual sense.

The objection might, however, be raised that not all the children lived to be as old as the parents were at the time said children were taken in charge, and that the parents may gradually have suffered a fall in the social scale, which may in the course of the years also happen to their children. The figures are consequently not directly comparable. Their main

value, however, is to show how many of the children from this group (I) — i.e. children taken in charge on account of unfortunate home conditions — became socially satisfactory as adults — in so far, that is to say, as a profession can be a criterion of this.

Summing up, it can, however, be said that rather over half of the boys and $^4/_5$ of the girls — as far as can be assessed by these investigations — found their place in society. Seen in the light of the situation during upbringing and the bad home conditions from which these children come, as also the hereditary tendencies which they possess, the result must be considered reasonably good.

PROGNOSIS OF SLIGHTLY DELINQUENT CHILDREN FROM BAD HOMES. (GROUP II)

This group consists of children who were taken in charge on a mixed indication, i.e. bad home conditions combined with slight signs of delinquency in the child itself. In some of these cases the home conditions were the determining cause for the intervention of the Board but at the time of being taken in charge the child showed slight signs of the onset of delinquency in the form of petty larceny, lying, other forms of untrustworthiness and so on. These children almost might be included in Group I but as the children showed the symptoms mentioned, it was thought to be more suitable to classify them in Group II. The great majority of this group, however, consists of children who themselves gave rise to the intervention of the Board by committing acts of delinquency which were, however, so slight in nature that they could not legally have been taken in charge for protective upbringing had not the home been found to be so bad as to render necessary the taking in charge of the child.

Of the original 166 boys and 75 girls who were included in this group 144 and 68 respectively are left to the estimate of the prognosis, making 86.7 and 90.6 per cent respectively of the total number in the group. We are now going to study the

development of these individuals after discharge on probation from foster-homes, children's homes or detention homes, beginning with the criminality aspect.

1. Prognosis as regards criminality.

After reaching their age of legal responsibility, almost every 3rd (43 cases) of the 144 boys were convicted of crimes registered in the Penal Register. The same applies to 6 of the women. The risk figures have been calculated in the usual way.

It is shown that for men the annual risk of being convicted of criminal actions is highest during the first 5 years and for these years as a whole is 5.3 per cent. The risk during the separate years of this first 5-year-period is 4.9, 4.9, 8.5, 4.6 and 2.9 per cent respectively. The frequency figures are not based on very large numbers, however, so that the differences between the different years are a little uncertain, though they suggest that the first years after discharge are the most critical.

After an observation period of 30 years, the total risk for boys is found to be 36.9 ± 6.9 per cent. Thus, if these men live at least 30 years after their discharge, rather more than $^1/_3$ will come to be punished.

Calculations have also been made of the risk at different years of age (see Table 56 b), which proved to be relatively evenly distributed over the different years before the age of 21. After this age, it decreases quite markedly.

A comparison with DAHLBERG's investigation (see p. 240), where there are special figures for Malmö, shows that the annual risk of crime in this group for the ages 15—18 years is not quite 4 times that of the normal figure, while for the 18—21-year-olds it is more than 10 times as much.

A comparison between the total risk for the total population and for the men in this group respectively shows that this is 8.4 per cent for the former, if they live to be 40, while it is more than 4 times higher (36.6 ± 5.8 per cent) for a man of the same age from Group II. The figure is not quite exact on account of the large standard error, but the difference from the normal figure is significant.

Table 56. **Average annual risk and calculated total risk in per cent for men of committing a crime, being reported to the Temperance Board because of inebriety, being warned or sentenced for vagrancy or of having to apply for poor relief. Group II.**

Table 56 a.

Years of obs. after discharge	Criminality	Age	Criminality
	Average annual risk		
1 – 5	5.3 ± 2.1	15—18	5.1 ± 2.2
6—10	1.3 ± 1.2	18—21	4.1 ± 1.9
11—15	1.9 ± 1.6	21—25	1.7 ± 1.4
16—20	—	25—30	1.3 ± 1.3
21—25	0.5 ± 1.1	30—35	0.3 ± 0.7
26—30	—	35—40	0.5 ± 1.1
		40—45	—
	Calculated total risk		
5	23.9 ± 4.8	18	14.5 ± 3.7
10	28.8 ± 5.2	21	24.6 ± 4.7
15	35.4 ± 5.8	25	29.6 ± 5.1
20	35.4 ± 5.9	30	34.1 ± 5.5
25	36.9 ± 6.1	35	35.1 ± 5 6
30	36.9 ± 6.9	40	36.6 ± 5.8
		45	36.6 ± 6.9

Table 56 b.

Age	Inebriety	Vagrancy	Poor relief
	Average annual risk		
15—20	0.2 ± 0.5	0.2 ± 0.4	0.9 ± 0.9
20—25	0.7 ± 0.9	0.5 ± 0.7	2.7 ± 1.5
25—30	1.1 ± 1.2	0.2 ± 0.5	4.3 ± 2.2
30—35	0.4 ± 0.8	0.3 ± 0.6	2.1 ± 1.8
35—40	2.1 ± 2.3	—	2.6 ± 2.3
40—45	·	—	—
	Calculated total risk		
20	1.0 ± 1.0	1.0 ± 0.9	4.5 ± 1.9
25	4.5 ± 2.2	3.5 ± 1.7	16.7 ± 3.8
30	9.6 ± 3.4	4.5 ± 2.0	33.1 ± 5.6
35	11.6 ± 3.8	6.0 ± 2.4	39.8 ± 6.2
40	20.5 ± 6.0	6.0 ± 2.8	47.2 ± 7.0
45	·	6.0 ± 4.3	47.2 ± 7.4

Table 57. **Average annual risk and calculated total risk in per cent for women of committing a crime, being warned or sentenced for vagrancy or of having to apply for poor relief. Group II.**

Years of obs. after discharge	Criminality	Age	Criminality	Age	Vagrancy	Poor relief
			Average annual risk			
1— 5 ...	0.6 ± 1.0	15—18 ...	1.5 ± 1.8	15—20 ...	0.8 ± 1.2	1.5 ± 1.7
6—10 ...	1.1 ± 1.4	18—21 ...	0.5 ± 0.9	20—25 ...	1.1 ± 1.4	0.4 ± 0.9
11—15 ...	—	21—25 ...	0.9 ± 1.3	25—30 ...	1.0 ± 1.6	1.4 ± 1.8
16—20 ...	0.6 ± 1.4	25—30 ...	—	30—35 ...	—	0.6 ± 1.4
21—25 ...	—	30—35 ...	0.6 ± 1.3	35—40 ...	1.0 ± 2.2	—
		35—40 ...	—			
			Calculated total risk			
5	3.0 ± 2.2	18	4.5 ± 3.2	20	4.0 ± 2.8	7.3 ± 3.8
10	8.2 ± 3.8	21	6.0 ± 3.5	25	9.1 ± 4.1	9.3 ± 4.2
15	8.2 ± 4.4	25	9.2 ± 4.2	30	13.6 ± 5.2	15.5 ± 5.5
20	10.8 ± 5.3	30	9.2 ± 4.8	35	13.6 ± 5.9	18.0 ± 6.1
25	10.8 ± 6.6	35	11.7 ± 5.5	40	17.8 ± 7.3	18.0 ± 7.1
		40	11.7 ± 6.8			

As regards the nature of the crime, 32 cases (i.e. 73 per cent) are seen to have committed crimes against property. Most of these consist of simple theft (16 cases); 6 were guilty of theft with house-breaking, and 10 of petty larceny. Of the remainder, 9 were sentenced for breach of military service, 1 for assaulting the police, and 1 for being drunk while driving.

As regards the level of intelligence. 26 were considered normal, while 8 were of dull intellect, and 3 feeble-minded. 6 were unknown quantities. Two had had their sentences remitted (5: 5), one on account of imbecility and one for no known reason.

The annual risk for *women* is 0.6 per cent for the first 5-year observation period, and 1.1 per cent for the second. (Table 57.) The difference may be due to random variation, as the material is small.

Here, as elsewhere, the annual risk of being sentenced for Penal Register crimes incurred by women is lower than for the men: cf. Tables 56 and 57. The figures for the sentenced cases are lower throughout, so that the general tendency can probably be considered certain, even if the difference between the different years may be attributable to chance. The same holds

if the risk is calculated according to years of age. The material is too small to establish whether there is any difference in annual risk between the ages of 15 and 25 years.

The total risk for women in this group is 9.2 ± 4.8 per cent at the age of 30 years: to judge from these somewhat uncertain figures, the risk figure for the women is about $^1/_4$ that for the men in the same group and of the same ages.

Compared with DAHLBERG's total figures for women during 1937, both the annual risk and the total risk for the women in this group is considerably higher than the average for criminal females. The total risk for town-dwellers of the former category at the age of 30 is 0.82 per cent, and for the latter, 9.2 per cent — that is to say, more than 10 times as much. The material is small, however, and the difference not significant.

For 4 girls, the crime was petty larceny, and for 2 embezzlement and fraud respectively. 4 were accounted intellectually normal, and 2 subnormal. Peculiarities of temperament were recorded for 1 case only.

It is also interesting to see how often there are relapses into crime. 19 of the 43 male offenders received a second sentence. 14 relapsed within 5 years, and not less than 10 of these during the first year after having served their sentence or, if they had been released on probation, in the year immediately following this. Thus, the risk is unquestionably highest during the first year. DAHLBERG and LINDBERG's figure for the total risk of relapse within 3 years after discharge from prison is 10 per cent, while ours is 32 ± 10 per cent i.e. about 3 times as high. A comparison with the 'normal figure' (28 per cent) for those sentenced to penal servitude yields no statistically significant difference, even if the figure is lower numerically.

The intelligence of the relapsing men was characterized as follows: 11 normal; 5 subnormal, 1 of these being feeble-minded; no data about the rest. Of the relapsing women, 2 were normal, 1 subnormal, and 1 not classified.

Of the 6 female first offenders, 4 were subsequently sentenced at least once. The figures are too small to allow of a closer analysis, however.

It would seem that children taken in charge due to bad home conditions and mild symptoms of delinquency incur

a greater risk of committing crimes and being sentenced according to the Penal Code, when adult, than does the population as a whole, as also that the risk is proportionately greater for the women of the first category than for the men (compared with the total population). And the risk of relapse in this collection of cases is greater than for criminals in general.

2. Prognosis as regards temperance.

The Temperance Board has taken action against not less than 6 men in this group.

As Table 56 b shows, the risk is highest for the ages 35—40 years. If these figures are compared with DAHLBERG's previously mentioned figures for Malmö (see p. 247), we find that the risk for the men in this group is 7 times the normal figure (0.3 per cent for men 1929—33). While the total risk for 40-year-old men from Malmö is 4.3 per cent, that for similar men from this group is 20.5 ± 6.0 per cent. That is to say, if the boys in this group live to the age of 40, there is a risk that $^1/_5$ of them have become alcohol addicts.

If we now study the incidence of fines for offences of drunkenness for reasons already mentioned taking only the persons born in 1917 and later, we find that, up to the age of 25 years, the total risk of at least one such fine is 19.5 ± 12.0 per cent. The corresponding figure for the men in the total population is 7.3 per cent (1938).

3. Prognosis as regards vagrancy.

A total of 6 men and 8 women from this group have been proceeded against on the score of vagrancy.

If, as before, the annual risk is calculated, it is found (see Table 56) that it is highest for the men between the ages of 20—25 years, i.e. 0.5 ± 0.7 per cent. The risk for the corresponding ages in the normal population in 1932 was 0.05 per cent. Similarly, the total risk for 40-year-olds in this group (6.0 ± 2.8 per cent) is 10 times higher than for men in the normal population, where the figure is 0.6 per cent. The large

standard errors mean that the differences are not significant, however.

The risk for women in this group is very much greater than that for the men (Table 57). The annual risk is almost as high at 15 as it is at 30 years of age, and is about 4—5 times higher than that for the men. The total risk at 40 years is 3 times that of the men, and 100 times that of women in the total population calculated for 1932 (i.e. 0.172 per cent). Thus, women in this group who reach the age of 40 run a great risk of vagrancy. Now, it should be noted that the term 'vagrancy' varies slightly according as it is applied to men or to women. Men addicted to this offence mainly belong to a group of indolent, unbalanced, sometimes itinerant, and more or less criminal individuals, whereas female vagrants are, in reality, prostitutes. The difference probably does not matter, since in both cases the individuals concerned are shirkers and parasites.

It may also be interesting to see what intellectual levels were recorded for these persons. Of the women, 3 were normal and 2 dull while 2 were feeble-minded. There were 4 normal and 2 subnormal men. A comparison between the sexes in this group thus shows that $^2/_3$ of the men but hardly $^1/_2$ of the women were of normal intelligence; however, the figures are of course too low to warrant any general assessment.

4. Prognosis as regards self-support.

A number of the individuals in this group have also had to apply to the Poor Relief Board for assistance, namely 50 men and 11 women.

These men are best off, relatively speaking, in the years immediately following discharge, and the risk is greatest 10—15 years after this date. If instead the years of age are considered the figures indicate that the risk of poor relief is highest in the ages between 25 and 30 years. Cf. Table 56. The total risk at the age of 40 years is 47.2 ± 7.0 per cent.

The risk figures for the women are fairly varied, due to the smallness of the material (see Table 57). There does not seem to be any one year where poor relief is most frequent. The total risk at 40 in this group is 18.0 ± 7.1 per cent. As already

mentioned, that for the men is 47.2 ± 7.0 per cent. The difference is statistically significant.

A closer analysis of the cases in receipt of relief shows that 20 of the men were granted minor assistance in some form, while 30 men needed more generous help. There was no record of any disabling disease against any of them, but 21 had been convicted of crimes. It is most likely that poor relief was extended to their families.

Summing up, we can say that, after the age of 40 years, the ability of this category to support themselves and their families is so reduced that $^1/_5$ of the women and almost half of the men have received public assistance in one form or another.

5. Social position.

In this group, too, it is interesting to compare the social position both with that of the total population and with that of the fathers. The same division as before has been used, and the calculation has in the same way only concerned those persons who were over 25 years of age when the investigation was ended.

As Tables 54 and 55 show, both the sons and the daughters as a rule came, when adult, to belong to the same social class as their fathers. The tendency to an increase in the percentual number in social class II is not statistically significant. As mentioned when group I was dealt with, the difference may also be no more than apparent, and spring from the change in the proportions of the social classes which has been taking place during latter decades.

On comparison, a greater number of both men and women in this group, are found to belong to social class III than is the case in the total population.

PROGNOSIS REGARDING MORE SERIOUS CASES OF DELINQUENCY. (GROUP III)

1. Prognosis as regards criminality.

Unlike the two foregoing groups, where bad home conditions were wholly or partially the cause of the children's deten-

tion, this group consists of children whose own character prompted intervention. It is true that a number come from bad homes, but quite a few are from tolerably good ones or from such which did not leave very much to be desired. The character of the home is discussed on page 156 and following.

601 boys and 238 girls, making 96.5 per cent and 96.4 per cent of the total number in the group, have been used for the prognosis. 222 boys and 31 girls (i.e. more than every 3rd boy and every 10th girl) were sentenced for crime when adult. We have calculated the annual risk in the usual way for a closer analysis of these figures.

The total risk of crime for the *men* up to and including the 5th year after discharge is 28.6 ± 2.6 per cent; that is to say, about every 4th boy was sentenced for crime of previously mentioned type within 5 years from his discharge (Table 58 a). The risk figure of 28.6 per cent is considerably higher than that which FRITZELL (1935) found in his investigation, namely 22.1 per cent, although this latter author's period of observation was somewhat longer (7 years). However, his figures are not based on risk calculations, and are therefore not fully comparable with ours.

A study of the risk of crime during the separate years of observation shows it to be somewhat over 7 per cent for each of the 3 first years; for the 4th and 5th it is not quite 5 per cent, and it remains fairly constant during the 6th—10th years at about 2.5 per cent.

The total risk calculated on years of observation is fairly high. If these men live and are kept under observation for 30 years after discharge, there is a risk that almost half of them (47.5 ± 3.6 per cent) will have been up against the law.

We have now tried to get an idea of the risk at the years after the discharge. Another way of looking at the problem is to try to get the risk figures for different years of age.

The annual risk of crime is found to be highest at the ages 15—21 years, when it is about 6 per cent, about the same for the different years; during the other ages before 25 it varies between 2.5 and 4.6 per cent.

A comparison of these figures with DAHLBERG's (1943) pre-

Table 58. **Average annual risk and calculated total risk in per cent for men of committing a crime, being reported to the Temperance Board because of inebriety, being warned or sentenced for vagrancy or of having to apply for poor relief. Group III.**

Table 58 a.

Years of obs. after discharge	Criminality	Age	Criminality
	Average annual risk		
1— 5	6.5 ± 1.2	15—18	6.0 ± 1.2
6—10	2.7 ± 0.9	18—21	6.2 ± 1.1
11—15	1.4 ± 0.7	21—25	3.0 ± 0.9
16—20	0.7 ± 0.6	25—30	1.4 ± 0.7
21—25	0.3 ± 0.5	30—35	1.0 ± 0.7
26—30	1.0 ± 1.3	35—40	0.4 ± 0.5
		40—45	1.0 ± 1.3
	Calculated total risk		
5	28.6 ± 2.6	18	16.9 ± 2.1
10	37.7 ± 2.9	21	31.4 ± 2.7
15	41.9 ± 3.1	25	39.3 ± 3.0
20	43.9 ± 3.2	30	43.3 ± 3.2
25	44.9 ± 3.3	35	46.1 ± 3.3
30	47.5 ± 3.6	40	47.1 ± 3.3
		45	49.6 ± 3.7

Table 58 b.

Age	Inebriety	Vagrancy	Poor relief
	Average annual risk		
15—20	0.4 ± 0.3	0.7 ± 0.4	1.1 ± 0.5
20—25	1.5 ± 0.6	0.5 ± 0.3	2.9 ± 0.8
25—30	1.6 ± 0.8	0.1 ± 0.2	4.1 ± 1.1
30—35	1.0 ± 0.7	0.1 ± 0.2	2.7 ± 1.1
35—40	1.1 ± 1.0	—	3.6 ± 1.5
40 – 45	1.0 ± 2.2	—	1.9 ± 1.6
	Calculated total risk		
20	2.0 ± 0.8	3.5 ± 0.9	5.5 ± 1.1
25	9.1 ± 1.6	6.0 ± 1.1	18.4 ± 2.0
30	16.2 ± 2.2	6.5 ± 1.2	33.8 ± 2.9
35	20.2 ± 2.6	7.0 ± 1.3	42.2 ± 3.3
40	24.5 ± 3.1	7.0 ± 1.3	51.9 ± 3.9
45	28.1 ± 4.9	7.0 ± 1.6	56.3 ± 4.2

viously mentioned figure for the total population in Malmö (see p. 240) shows the annual risk of crime to be about 4 times higher in our material for the age group 15—18 years, and up to about 20 times higher for the age group 18—21 years (6.2 as against 0.34 per cent); for the following years it is on an average about 10 times higher than the risk among the population of the town.

The total risk calculated up to different ages is also high. If these men live to be 45, one half (49.6 ± 3.7 per cent) have been found guilty of crime. As the total risk of crime for 45-year-olds among the total population of Malmö is 8.8 per cent, it means that the total risk for men belonging to our group is more than 5 times higher. The material in this group is comparatively large, so that the differences we have mentioned are statistically significant.

The annual risk of crime for the *women* varies irregularly during the first 10 years after discharge from 1 to 3.6 per cent. It is therefore impossible to say which of these 10 years is most fraught with risk.

For the first 10 years after the discharge the total risk is rather over 15 per cent (Table 59), rising slightly during the next 5-year-period, and afterwards remaining fairly constant. That is to say, most of the girls in this group who commit crimes when adult do so within 10 years after discharge. If they have escaped comment up to this time, the risk of crime is very small.

Our total risk figure up to 5 years after discharge, 10.1 ± 2.3 per cent, is thus rather higher than that of AHNSJÖ for the same period of observation, i.e. 7 per cent. The difference may be due to chance.

The annual risk of crime is fairly irregular for women for the ages 15 to 25 years, varying from 0 for 15—16 years of age, to 3.1 per cent for 21—22-year-olds, where it is consequently highest.

At the age of 40 years, the total risk for these women is somewhat over 20 per cent; that is to say, at this age, every fifther woman showing delinquent symptoms of a graver kind when a child, had been sentenced for a fairly serious crime.

Table 59. **Average annual risk and calculated total risk for women of committing a crime, being warned or sentenced for vagrancy or of having to apply for poor relief. Group III.**

Obs. years	Criminality	Age	Criminality	Age	Vagrancy	Poor relief
			Average annual risk			
1—5.....	2.1 ± 1.0	15—18 ...	2.2 ± 1.7	15—20 ...	1.0 ± 0.9	2.1 ± 1.3
6—10 ...	1.2 ± 1.0	18—21 ...	1.5 ± 0.9	20—25 ...	1.6 ± 0.9	3.7 ± 1.4
11—15 ...	0.4 ± 0.7	21—25 ...	1.7 ± 1.0	25—30 ...	0.7 ± 0.8	1.4 ± 1.1
16—20 ...	—	25—30 ...	1.1 ± 1.0	30—35 ...	0.3 ± 0.6	0.8 ± 1.1
21—25 ...	—	30—35 ...	—	35—40 ...	—	1.0 ± 1.6
		35—40 ...	—			
			Calculated total risk			
5	10.1 ± 2.3	18	6.5 ± 2.9	20	5.0 ± 2.1	10.1 ± 3.0
10	15.4 ± 3.0	21	10.7 ± 3.3	25	12.3 ± 2.9	25.6 ± 4.1
15	16.9 ± 3.2	25	16.6 ± 3.7	30	15.3 ± 3.3	30.6 ± 4.5
20	16.9 ± 3.5	30	21.1 ± 4.2	35	16.8 ± 3.5	33.3 ± 4.8
25	16.9 ± 4.1	35	21.1 ± 4.3	40	16.8 ± 4.0	36.6 ± 5.3
		40	21.1 ± 4.7			

If we compare the annual risk figure (2.2 ± 1.7 per cent) for women between 15—18 years old in this group with the number of female first offenders of the same age from all the towns in Sweden in 1937 (see Table 49), which is only 0.065 per cent, we find our group leading by almost 30 times. The material of delinquent girls at our disposal is small, however, and more is required to assess the magnitude of the difference.

A comparison between the risk of crime for men and women in this group shows the annual risk for the women during the first 5 years of observation to average $^1/_3$ that for the men. This is quite a high proportion when we remember that, according to DAHLBERG (1943), the same figure for the women in the total population was $^1/_{10}$ that for the men in the year of 1937.

Most of the first crimes committed by the men were, as usual, crimes against property, which make up 78.9 per cent, distributed over simple theft, housebreaking, and more or less equal incidence of embezzlement and forgery. After this come

breach of military service, mainly truancy. Maltreatment and violence occur to about 5 per cent (12 cases). There are two cases of arson.

Sexual crimes are relatively few (4 cases). Other crimes are of various types, such as illicit 'borrowing', drunkenness while driving, etc.

For the women, too, most of the crimes were crimes against property, i.e. in 78 per cent (25 cases), of which simple theft was the commonest. Sexual offences, such as abortion and breaches of the *lex veneris,* were found in somewhat over 15 per cent (5 cases).

As regards the intellectual level of these children, most of the men had been denoted of at least normal intelligence (132 cases), 38 were recorded as poorly endowed, and 12 as feeble-minded. Thus, while over 70 per cent possessed normal gifts, about 20 were subnormal, and only about 5 per cent belonged to the category considered ripe for a special class. There is no data as to 40 cases. That is to say, the feeble-minded constitute little more than 5 per cent of the entire material, which should be the proportion they form of the population as a whole. Compared with DAHLBERG's figure of 3 per cent for children belonging to special classes in Stockholm, however, our figure is somewhat higher (see Chapter XI).

Risk of relapse.

Proceeding from the figures of DAHLBERG and LINDBERG (1944), we would expect the risk of relapse for the men should be at least 10 per cent and at most about 30 per cent within 3 years from the first crime, according to the nature of the first sentence meted out, these authors' work showing as it did that the risk of relapse was not at all the same after imprisonment as after penal servitude; actually this means that the risk of relapse depends on the gravity of the first offence.

Of the 222 men in this group committing a first crime, 109 relapsed within 20 years — i.e. in round numbers, not quite half. The annual risk of relapse is highest during the 1st and 2nd year after serving sentence for the first crime, being 27

and 18 per cent respectively. Almost $^3/_4$ of the relapses took place within 5 years from the first crime.

No classification into imprisonment or penal servitude has been made of the sentences in this material, and the figures are therefore not directly comparable with those of DAHLBERG and LINDBERG; nevertheless, the risk of relapse must be regarded to be on the high side.

The total risk of renewed crime for a man in this group is thus fairly high, reaching 65.0 ± 7.5 per cent after 15 years of observation (see Table 52). The material for subsequent years is too small to allow of conclusions.

The risk of relapse for women is considerably lower. Only 1 of the 31 first offenders received a second sentence.

The intellectual level of the relapsing cases was recorded as normal in 65 cases; 18 were poorly endowed, while 5 were feeble-minded and there were no data about 21. We can therefore say that over half were considered to be normally equipped, and that only rather over $^1/_5$ (23 cases) were subnormal, of whom 5 were feeble-minded, i.e. 4.5 per cent of the total number of relapsing cases. The figure approximates to the number of feeble-minded in the population.

Half of the first offenders subsequently sinned again, some of them more than once; these can therefore be denoted frequent offenders. A special survey of these shows that half (30 cases) were reputedly of normal intelligence, $^1/_3$ poorly equipped, and 3 cases feeble-minded. Unfortunately, there is no information about 14 cases. The distribution therefore seems to be the same as above. It does not seem, therefore, as if the worst equipped should be those who join the ranks of the old offenders.

2. Prognosis as regards temperance.

The addiction of many of the males in this group to alcohol should appear from the large number who have incurred measures from the Temperance Board during the course of the years, i.e. a total of 72 of 486 men born 1900 and later. With the exception of the youngest age-group 15—20 years, the

annual risk is about the same for all ages. (Table 58 b). A comparison of the annual risk figures with those of DAHLBERG (1942) for Malmö for 1929—1933 show those for the men of this group to be considerably higher than for the male population in general; the total risk at the age of 45 years is 28.1 ± 4.9 per cent, whereas the corresponding figure for the men in Malmö (1929—33) was 6.2 per cent.

3. Prognosis as regards vagrancy.

In this group, 31 men and 25 women have been sentenced to the work-house or cautioned, or — in the case of the younger ones — been taken in charge by the Child-Welfare Board on the score of indolent or vicious living (§ 22 d).

The annual risk for the men is highest between the ages of 15—20 years (Table 58 b). Our figure is at least 20 times higher than that of DAHLBERG and LINDBERG for these ages in the total population, which for 1932 was 0.03 ± 0.04. The total risk for men in this group who live to be 45 years of age is 7.0 ± 1.6 per cent, i.e. 10 times higher than the normal value (0.72 per cent).

For the women in this group, the annual risk is highest between 20—25 years of age, when it is 1.6—0.9 per cent — that is to say, considerably higher than DAHLBERG and LINDBERG's figure for 1929, which for this age-group was only 0.017 per cent. The total risk at 40 is 16.8 ± 4.0 per cent, also appreciably higher than the normal value of 0.17 per cent. The risk that gravely delinquent girls will, when adult, be addicted to vagrancy is thus about 100 times greater than the one to which other girls are exposed. It is much more usual for the women belonging to this group to fall foul of the vagrancy laws than for the men — to judge from the risk figures, at least twice as usual. As already mentioned, the term 'vagrancy' does not mean quite the same thing for men as for women, for the latter being another word for prostitution. (Table 59.)

4. Prognosis as regards self-support.

It is plain that a number within this group have to rely on public assistance for their means of support. Not less than over

$^1/_3$ of the males (219 cases) and $^1/_4$ of the females (59 cases) have been so badly off financially that they have had to apply to the Poor Relief Board for aid, at any rate of a temporary nature. As already mentioned, this applies to all cases — not only more permanent aid but also that of a temporary kind, for example in sickness, and occasional allowances to wife and children, so that these figures can be accounted maxima. We may add that about $^1/_3$ of the cases concerned received aid of the latter kind, while almost $^2/_3$ of the cases required relief of a more permanent kind. For the men the annual risk is highest at the ages 25—30 years, then being 4.1 ± 1.1 per cent (Table 58 b). The total risk for 40-year-old men, i.e. 51.9 ± 3.9 per cent, shows that over half the men of this age risk poor relief at least once. For the women the risk is highest between the ages of 20 and 25 years — that is to say, earlier than for the men in the same group. (Table 59). At the age of 40, the total risk is 36.6 ± 5.3 per cent, at least $^1/_3$ of the women thus risking social assistance at this age. Rather more men than women seem to apply for public aid in this group.

5. Social position.

It may also be worth seeing the callings which these children adopt as adults, thus getting an idea of their social position. To judge from the foregoing investigation, we would hardly expect them to have reached any very high social level.

A comparison with the normal population shows that considerably more of the men in this material belong to social class III than would have been expected, and that social class II has been achieved by not even half as many as ought to have belonged there. (Table 54). A comparison with the position of the parents shows that about the same number of sons as fathers can be relegated to class II, but that a smaller number of sons belong to class III, whereas more sons than fathers have joined class IV. Clearly, then, the sons in this group are socially lower than their fathers. As in the foregoing groups, several girls can be seen to have outstripped their fathers and got into class II.

probably mainly by marriage. A rather larger percentage have sunk into the lowest social class, however — that is to say, the class of the more uncertain occupations.

PROGNOSIS AS REGARDS CRIMINAL JUVENILES. (GROUP IV)

1. Prognosis as regards criminality.

As pointed out in the Introduction, the juveniles in this group are to be considered as having already offended once on their inclusion in the material. They are thus to be accounted to a certain extent as relapsing cases. Actually, however, they are on a par with most of the boys in Group III (graver cases of delinquency), who often have been guilty of the same kinds of dereliction. The only aspects in which these groups differ concern the authorities stepping in and, to a certain extent, the age at which the action was performed. It may therefore be questioned whether a comparison should be made with normal figures for relapse, or whether this group should, like the other groups, be compared with the figures for first offenders in general. However, since this group has been selected with regard to committed crimes which have been taken to court, it may be most correct to compare a first new crime with the figures for relapsing cases; a comparison should also be made with first offenders, however.

After adding the material from both periods, the prognosis includes 681 men and 51 women, making 98.4 per cent and 96.2 per cent respectively of the primary material. Of these, 226 men and 12 women have relapsed into crime.

The annual risk of the first new crime is found to be highest between the ages of 15—18 years, as is nowadays also the case for first offenders in general (DAHLBERG 1943). The annual risk is 11.4 ± 2.1 per cent, which is nearly 10 times higher (Table 60 a) than the normal figure for first offenders of the same age in Malmö, i.e. 1.3 per cent. The total risk calculated on years of age is also very high, in that more than half (52.4 ± 4.2

Table 60. **Average annual risk and calculated total risk for men of committing a crime, being reported to the Temperance Board because of inebriety, being warned or sentenced for vagrancy or of having to apply for poor relief. Group IV.**

Table 60 a.

Obs.-years	Criminality (Crime No. 2)	Age	Criminality (Crime No. 2)
	Average annual risk		
1—5	6.4 ± 1.0	15—18	11.4 ± 2.1
6—10	1.3 ± 0.6	18—11	5.4 ± 1.0
11—15	1.0 ± 0.6	21—25	1.9 ± 0.7
16—20	0.3 ± 0.4	25—30	1.2 ± 0.6
21—25	0.8 ± 0.7	30—35	0.4 ± 0.4
26—30	0.4 ± 0.9	35—40	0.6 ± 0.6
31—35		40—45	0.6 ± 0.8
	Calculated total risk		
5	28.1 ± 2.3	18	30.4 ± 3.7
10	32.6 ± 2.5	21	41.2 ± 3.9
15	36.0 ± 2.7	25	45.6 ± 3.9
20	37.0 ± 2.7	30	48.7 ± 4.0
25	39.5 ± 2.9	35	49.7 ± 4.0
30	40.5 ± 3.2	40	51.2 ± 4.1
35	·	45	52.4 ± 4.2

Table 60 b.

Age	Inebriety	Vagrancy	Poor relief
	Average annual risk		
15—20	1.4 ± 0.7	0.4 ± 0.3	1.2 ± 0.6
20—25	1.2 ± 0.5	0.4 ± 0.3	2.9 ± 0.7
25—30	0.9 ± 0.6	0.3 ± 0.3	2.5 ± 0,8
30—35	0.9 ± 0.7	0.1 ± 0.2	2.7 ± 1.0
35—40	0.9 ± 0.8	0.2 ± 0.3	2.8 ± 1.3
40—45	0.9 ± 1.4	—	3.8 ± 2.1
	Calculated total risk		
20	6.8 ± 1.5	2.0 ± 0.7	5.9 ± 1.3
25	12.3 ± 1.8	4.0 ± 0.9	18.8 ± 2.0
30	16.3 ± 2.1	5.5 ± 1.1	28.4 ± 2.5
35	19.9 ± 2.5	6.0 ± 1.2	37.5 ± 3.0
40	23.4 ± 2.9	7.0 ± 1.3	45.8 ± 3.5
45	26.9 ± 3.7	7.0 ± 1.6	55.4 ± 4.3

per cent) of the men in this group who reach the age of 45 years run the risk of committing at least one crime — i.e. about 6 times the figure for the normal population (8.8 per cent).

However, if the figures are calculated on the period of observation, the total risk of relapse into crime in the first 3 years after sentence of probation or fines, or within 3 years after serving imprisonment, is 22.6 ± 1.8 per cent. This figure is somewhat lower than the previously cited total figure (24.6 per cent) given by DAHLBERG and LINDBERG (1944) for relapse within 3 years for youths who, at their first prison sentence, were 15—20 years of age — i.e. the same ages as the youths in this group.

When making a comparison, we must of course take into account the type of sentence which these youths incurred the first time. A review (see Chap. XII) shows that most of the cases — almost $^3/_4$, i.e. 72.3 per cent — were fined or released on probation (500 of 692). Only 2.9 per cent (20 cases) were sentenced to imprisonment, and 12.3 per cent (85 cases) to penal servitude. For the sake of completeness, it may be added that a further 12.3 per cent were committed to reformatories, and that 2 had their sentence remitted on mental grounds.

After 30 years of observation, the total risk amounts to 40.5 ± 3.2 per cent; that is to say, a good one third of those committing a first crime proceed to a second. (Table 60).

The women in this group are, actually, too few (51 cases), and the risk of relapse in women too small, to allow of any more general conclusions. However, as for the men, the annual risk of relapse into crime is found to be highest in the first 5 years after the first offence, and highest of all in the first year; as for the youths, it is greatest in the ages between 15—18 years (Table 61). The total risk of relapse for a female first offender is 27.0 ± 8.2 per cent within 15 years; thus, more than every 4th woman risks relapse within this period.

It may then be interesting to study if, and to what extent, these juveniles continue to sin — i.e. commit a third crime.

111 of the 226 relapsing male offenders commit fresh crimes. To recapitulate: 226 of the 681 first offenders (i.e. about $^1/_3$) have relapsed once. Of these 226, about half have committed a

Table 61. **Average annual risk and calculated total risk in per cent for women of committing a crime or of having to apply for poor relief. Group IV.**

Obs. years	Criminality (Crime No. 2)	Age	Criminality (Crime No. 2)	Age	Poor relief
		Average annual risk			
1— 5 ...	4.7 ± 3.2	15—18 ...	7.1 ± 6.7	15—20 ...	1.6 ± 2.6
6—10 ...	0.6 ± 1.4	18—21 ...	5.1 ± 3.5	20—25 ...	2.0 ± 2.2
11—15 ...	0.8 ± 1.8	21—25 ...	1.4 ± 2.0	25—30 ...	1.3 ± 2.0
16—20 ...	—	25—30 ...	0.7 ± 1.6	30—35 ...	—
21—25 ...	—	30—35 ...	—	35—40 ...	3.6 ± 4.5
		35—40 ...	—		
		Calculated total risk			
5	21.5 ± 7.2	18	19.8 ± 11.9	20	7.7 ± 5.7
10	24.0 ± 7.6	21	31.5 ± 12.8	25	16.5 ± 7.3
15	27.0 ± 8.2	25	35.1 ± 13.1	30	21.8 ± 8.2
20	27.0 ± 9.1	30	37.3 ± 13.3	35	21.8 ± 8.9
25	27.0 ± 10.8	35	37.3 ± 13.7	40	34.8 ± 11.9
		40	37.3 ± 14.2		

further crime (number 3), and can thus be designated relapsing offenders.

The risk of a third crime (or a *second* relapse) is also highest within the first 5 years, the total risk here being 55.1 ± 7.0 per cent. (Table 52). This means, then, that a good half of the second offenders relapse. The risk is highest of all during the first year, where it is 24 per cent; i.e. one quarter relapse. In the 2nd year the risk is 14 per cent, and in the following 3rd—5th years 10 per cent. The total risk of a 3rd crime within 15 years is 65.4 ± 7.6 per cent.

There is not really anything much to add as to the type of crime of which the men have been guilty. Crimes against property amount, in round numbers, to 80 per cent, sexual offences to 3 per cent, and violence and maltreatment to 8 per cent.

If, using the available data in records etc., we try to assess the intelligence of these relapsing offenders, we find that over half are of normal powers i.e. 58 per cent (or 131 cases), 16 per cent weakly endowed (37 cases), while 13 per cent (30 cases)

have been denoted feeble-minded, and 28 have no note against them. These latter constitute a considerably higher percentage compared with previous groups. Seeing that 9 per cent of the total number of boys in this group were considered feeble-minded, it seems as though the cases with the weakest intelligence showed the greatest tendency to relapse into crime.

Relatively few women go as far as committing a 3rd crime and being sentenced for it, though 6 of the original 51 have done so. These, too, show the same tendency as the men: namely, the greatest risk in the year immediately following the previous crime. The material is too small to warrant any further conclusions.

Anyhow, it is of a certain interest to see how the risk of a first relapse stands with regard to the different measures meted out, i.e. probation, reformatory, prison, or penal servitude.

As already pointed out, most of the youths (500 of 692) were given a mild sentence, in the form either of fines or of probation, which of course in some cases also consisted of fines. Of these 500, 164 have subsequently offended again. As before, the total risk has been calculated, amounting to 28.1 ± 2.7 per cent for the first 5 years, and 40.5 ± 3.6 per cent after 25 years of observation. (Table 62).

Committal to a reformatory must be taken to be the next mildest sentence. Of the 85 youths thus detained, 27 have subsequently relapsed into crime: more correctly, 26.6 ± 6.4 per cent have relapsed within 5 years. The total risk in this category after an observation period of 25 years is 36.9 ± 8.5 per cent, however.

For 105 youths and 3 girls, the first sentence consisted of imprisonment or penal servitude. Of these cases, 35 men and 2 women have afterwards relapsed. The total risk for the men during the first 5 years of observation is 28.6 ± 6.2 per cent, and 39.3 ± 7.4 per cent after 25 years. Thus, in round numbers, $^1/_4$ of these have also relapsed within 5 years.

There therefore seems to be hardly any difference in the risk of relapse between those cases incurring sentence of different intensity: largely speaking, rather more than $^1/_4$ of the youths

Table 62. **Average annual risk and calculated total risk in per cent of a first relapse into crime (crime No. 2) for men, distributed according to the penalty imposed for crime No. 1. Group IV.**

Years after the first crime	Fine or probation	Reformatory education	Imprisonment or penal servitude
	Average annual risk		
1— 5	6.4 ± 1.2	6.0 ± 2.8	6.5 ± 2.8
6—10	1.3 ± 0.7	0.4 ± 0.9	2.5 ± 2.1
11—15	1.0 ± 0.7	1.6 ± 2.1	0.4 ± 0.9
16—20	0.4 ± 0.5	—	—
21—25	1.0 ± 1.1	1.0 ± 2.3	0.4 ± 1.2
	Calculated total risk		
5	28.1 ± 2.7	26.6 ± 6.4	28.6 ± 6.2
10	32.6 ± 2.9	28.1 ± 6.5	37.1 ± 7.0
15	36.0 ± 3.1	33.7 ± 7.3	38.3 ± 7.1
20	37.5 ± 3.2	33.7 ± 7.8	38.3 ± 7.2
25	40.5 ± 3.6	36.9 ± 8.5	39.3 ± 7.4

have relapsed within 5 years after the first crime. We would have expected those receiving the mildest sentence to have shown a better prognosis than the others, but this has plainly not been so. But no more must be read from the figures than what they actually show. It is not certain that the above categories represent different degrees of offence. It is conceivable, though not likely, that those committed to reformatories were graver cases but that their risk of relapse was reduced thanks to the measures taken. Without a thorough investigation it is impossible to pass any verdict on the implications of the figures, beyond the purely factual one that the difference between the groups under the conditions then prevailing is not great.

2. Prognosis as regards temperance.

The investigation into the addiction of the men in this group to alcohol, and their consequent embroilment with the Temperance Board, has shown that at least 79 of 571 born 1900 and later can be considered inebriates. The risk is about the same for all ages between 15 and 45 years (Table 60 b). The total risk at the age of 45 years is 26.9 ± 3.7 per cent, and is

about 4 times as high as for the Malmö men in general of this age. As mentioned before, the figure is a minimum.

If, however, the risk of sentence for drunkenness is calculated for the younger men (born 1917 or later), the annual figure is found to be relatively high for this group, i.e. 3.7 ± 1.7 per cent for 15—20-year-olds, and 2.5 ± 1.5 per cent for 20—25-year-olds. The total risk for 20- and 25-year-olds is 17.2 and 27.1 per cent respectively. Compared with DAHLBERG's (1943) total risk figures for the normal population in 1938 — the year with the lowest normal figures — of 2.1 and 7.5 per cent respectively for the same age groups, the results for the younger ages in the present group are surprisingly high: 8 times higher for 20-year-olds and 4 times higher for 25-year-olds.

This does not necessarily mean that the corresponding figures for the older ages in this group would be much better. If it had been possible to obtain uniform and comparable figures for these latter, it is very likely they would have been at least as high, and perhaps higher.

3. Prognosis as regards vagrancy.

Needless to say, a number of the juveniles in this group have also been addicted to vagrancy, but only 27 men and 4 women have actually fallen foul of the vagrancy laws. The annual risk is highest in the first 5 years after sentence or discharge from a corrective institution, and highest of all in the first 3, averaging 0.5 per cent. Calculated according to age, the risk is greatest between 15—20 years. (Table 60 b). The total risk for men reaching the age of 45 is 7.0 ± 1.6 per cent. A comparison of these figures with the normal figures shows the annual risk to be about 10 times that for ordinary vagrants at the age of 20 years, which last is 0.04 per cent. The total risk at 45 years of age (7.0 per cent) is about 10 times higher than the normal figure for this age (0.7 per cent).

Very few of the women in this group have come up against the vagrancy laws: 4 out of 51 (see above). All 4 were 20—25 years old. The material is too small to allow of any calculations of risk; the figures are mentioned only for the sake of completeness.

4. Prognosis as regards self-support.

The next question to present itself concerns the financial position of these individuals when adult. As before, this question can hardly be answered except by studying how many of these persons were in so bad a way that they had to apply for poor relief. 221 of the men and 12 of the women are found to have been in receipt of relief. As before, these numbers must be regarded as maxima, since even temporary help has been included. The annual risk for the men in this group is, on an average, highest at the ages of 40—45 years, where it is 3.8 ± 2.1 per cent (Table 60 b). The same risk sets in earlier for the women, namely at the ages of 35—40 years, where it is 3.6 ± 4.5 per cent (Table 61). The figures are not definite, however; a better view of the extent of relief granted to the individuals of this group is obtained if the total risk of relief at different years of age is studied. It is found that almost half, i.e. 45.8 ± 3.5 per cent, of the men, and a third of the women, 34.8 ± 11.9 per cent, have been in receipt of public assistance by the age of 40.

5. Social position.

62 per cent of the men in Malmö belong to social class III. The figure in our material is 83.2 per cent (if classes III and IV are made into one). A similar difference is obtained for the women: 57 per cent for the population in 1940, and 80.6 per cent in our material. Social class II is considerably less represented by either sex in our material than in the population.

If the social position of the sons is compared with that of their fathers, we notice that several of the sons have remained in the more uncertain occupational group which class IV must be considered to constitute, and that consequently a fewer number is found in class III. On the other hand, the same number of sons as fathers belong to class II. The girls have scarcely shown any advance in this group, their social position agreeing on the whole with that of their fathers (Tables 54 and 55).

CHAPTER XIV.

COMPARISON BETWEEN THE DIFFERENT GROUPS FROM PROGNOSTIC ASPECTS.

In foregoing chapter the prognosis has been followed from different aspects in each group separately. It may now be interesting to compare the various groups with each other as regards these different aspects.

First, then, there is the risk of sentence for criminal actions, of the kind registered in the Central Penal Register, which were committed after discharge from institution or private home.

A comparison between the different groups shows that, for the *men*, both the annual figures and the total risk of being sentenced for a first offence are not the same in all, and are consistently lowest for Group I and highest for Group IV, if the latter cases are put on a par with first offenders. This can also be seen from Fig. 11, which illustrates how the different groups stand in respect of the total risk of crime, compared with the total risk for the male population of Malmö in 1937. The curve for men belonging to Group I is lowest, and comes nearest to DAHLBERG's curve for the total risk of crime for men in Malmö, though it is considerably higher than this latter. The curve for Group II is higher than that for Group I, while those for Groups III and IV reach considerable heights, particularly that for Group IV.

If we compare the different groups as regards the risk of relapse, we find (Table 52 a, p. 243) it is highest for III and IV, and that these groups are about equal in this respect, if crime 3 in Group IV is equated to crime 2 in Group III.

Fig. 13 shows the total risk of incurring measures from the Temperance Board in all groups. For the younger cases, the

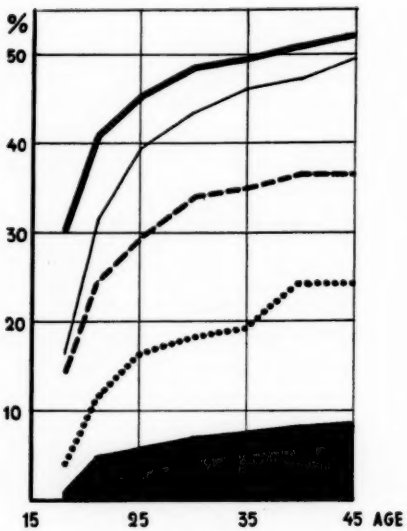


Fig. 11: Men.

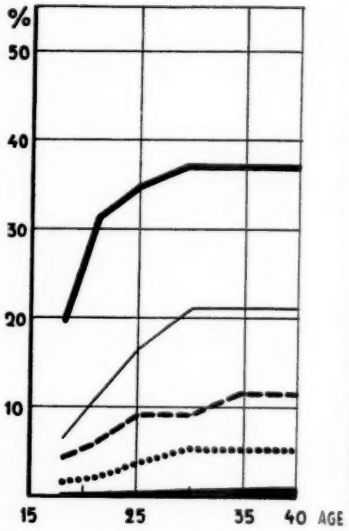


Fig. 12: Women.

Figs. 11 and 12. Calculated total risk in per cent of being sentenced for crimes recorded in the Central Penal Register for men and women in different ages in Groups I—IV and for the total male population of Malmö in 1937, respectively for women in the corresponding ages in the towns of Sweden in 1937.

Group I: dotted line, group II: broken line, group III: whole fine line, group IV: whole thick line. The black segment indicates the total male, respectively female, population.

total risks are low, and about the same for Groups I, II, and III. This is probably because, while young, these individuals are still under supervision; this means that they either have less chance to offend in this way, or — if their offences are of a more serious nature — that they are more liable to renewed detention than to action from the Temperance Board. For the older cases — men who have reached the ages of 35—40 years — the risk increases in all groups, but there is a plain difference between Groups I and III, the risk in the latter being nearly twice as high as in the former (total risk at 35 years being

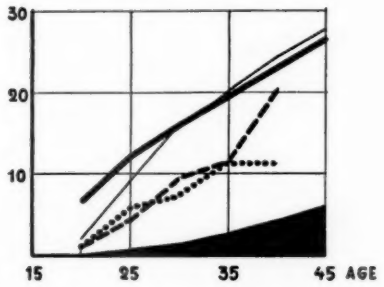


Fig. 13. Calculated total risk in per cent of being reported to the Temperance Board because of inebriety for men born in 1900 or later in groups I—IV and for the total male population of Malmö 1929—1933 in the corresponding ages.
Group I: dotted line, group II: broken line, group III: whole fine line, group IV: whole thick line. The black segment indicates the total male population.

11.4 ± 3.8 per cent as against 20.2 ± 2.6 per cent). Group IV yields the highest risk figure, even for the younger cases.

As regards the men, the risk of being sentenced for drunkenness in younger years is highest for the criminal youths (Group IV); the total risk at 20 years of age is nearly 7 times, and at 25 years of age more than twice, that in Group I. Thus, it is much more usual to find addiction to alcohol among young men who have committed crimes than among those who have merely come from bad homes.

Fig. 14 shows that Group I presents no great difference from the others as regards the risk run by men of coming up against the vagrancy laws. Both the annual and the total risk for all groups is low throughout, though it is considerably higher than the normal figure.

Fig. 16 shows that Group I has the fewest men who were forced to apply for poor relief, and Groups III and IV the most. The differences between the total risk in each group are not great, however; the greatest difference is found between Groups I and III for the age of 40 years, where it is 14.7 ± 6.7 per cent.

Nevertheless, it is very likely that these risks are to a greater

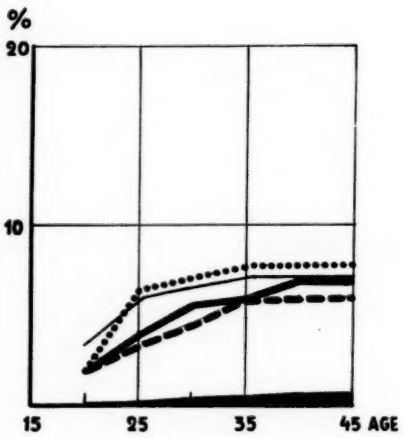


Fig. 14: Men.

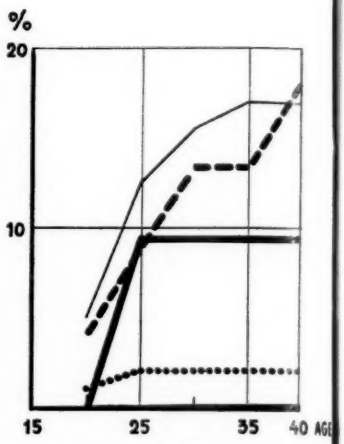


Fig. 15: Women.

Figs. 14 and 15. Calculated total risk in per cent of being warned or sentenced to compulsory labour because of vagrancy for men and women in different ages in groups I—IV and for the total male and female population in Sweden 1932.

Group I: dotted line, group II: broken line, group III: whole fine line, group IV: whole thick line. The black segment indicates the total population and is for the women hardly visible in the scale used, which is double that used in Figs. 11—12 and 16—19.

or lesser extent correlated — that is to say, that they have often overtaken one and the same person. It is therefore interesting to see how many persons have escaped any kind of action.

The calculations made of the chance of avoiding intervention by the public do not include measures dictated by inadequate powers of self-support, however. The reason for this is that poor relief e.g. during disease is a very common phenomenon in the social class to which the persons of this investigation belong, and disease anyhow does not constitute any real indication of social failure.

A survey of measures taken by the public as a result of more serious crimes (»Penal Register crimes»), vagrancy or inebriation, among men, shows (see Table 63 and Fig. 18 p. 285) that Group I unquestionably had the best prospects, while the other

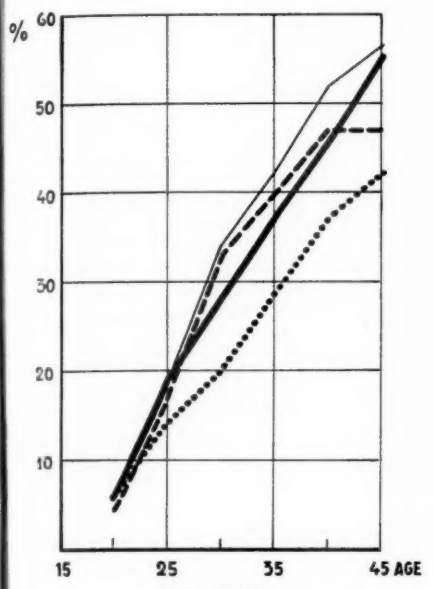


Fig. 16: Men.

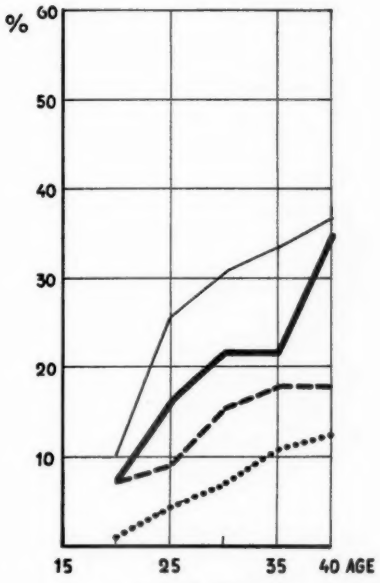


Fig. 17: Women.

Figs. 16 and 17. Calculated total risk in per cent of having to apply for poor relief for men and women in different ages in groups I—IV. Group I: dotted line, group II: broken line, group III: whole fine line, group IV: whole thick line.

groups had the worst. These last three seem pretty much on a level. The men once taken in charge by the Child-Welfare Board for prophylactic ends (Group I) yield, at 40 years of age, a figure of 69.7 ± 5.2 per cent for the total prospect of avoiding measures of any kind. That is to say, as far as these investigations show, a good two-thirds have been able to take their place in society. Group II (children taken in charge on mixed indications) have rather worse prospects, in that by the age of 40 years only 48.6 ± 8.4 per cent — i.e. not quite half — have steered clear of any contact with the authorities. The prospects of Groups III and IV (delinquent and criminal boys respectively) are somewhat worse at this age, only about

Table 63. Average annual prospect and calculated total prospect in per cent for men born in 1900 or later of escaping action by the society because of serious crimes, inebriety or vagrancy.

Age	Group I	Group II	Group III	Group IV
	Average annual prospect			
15—20	98.0 ± 1.1	94.7 ± 2.4	92.4 ± 1.5	90.5 ± 1.7
20—25	97.5 ± 1.4	97.3 ± 1.9	95.2 ± 1.3	96.5 ± 1.0
25—30	99.0 ± 1.0	97.5 ± 2.1	97.7 ± 1.1	98.4 ± 0.8
30—35	99.5 ± 0.8	99.5 ± 1.1	98.9 ± 0.9	99.7 ± 0.9
35—40	98.9 ± 1.7	96.9 ± 3.5	99.2 ± 1.0	98.2 ± 1.1
	Calculated total prospect			
20	90.4 ± 2.4	76.1 ± 5.4	67.3 ± 3.4	60.7 ± 3.9
25	79.7 ± 3.7	66.4 ± 6.3	52.6 ± 3.9	50.8 ± 4.1
30	75.7 ± 4.1	58.5 ± 7.0	46.8 ± 4.1	46.8 ± 4.2
35	73.7 ± 4.3	57.0 ± 7.2	44.3 ± 4.2	43.8 ± 4.3
40	69.7 ± 5.2	48.6 ± 8.4	42.6 ± 4.4	41.3 ± 4.5

Table 64. Average annual prospect and calculated total prospect in per cent for women, born in 1900 or later, of escaping action by the society because of serious crimes, inebriety or vagrancy.

Age	Group I	Group II	Group III	Group IV
	Average annual prospect			
15—20	99.5 ± 0.6	98.3 ± 2.2	97.2 ± 1.7	92.4 ± 6.2
20—25	99.7 ± 0.5	99.0 ± 1.6	98.0 ± 1.1	97.9 ± 2.7
25—30	99.6 ± 0.6	—	98.9 ± 1.1	99.1 ± 2.1
30—35	—	99.0 ± 2.2	—	—
	Calculated total prospect			
20	97.5 ± 1.3	91.8 ± 4.8	86.8 ± 3.8	67.4 ± 14.0
25	96.0 ± 1.6	87.3 ± 5.8	78.5 ± 4.4	60.6 ± 14.6
30	94.0 ± 2.1	87.3 ± 6.6	74.3 ± 4.8	58.1 ± 14.8
35	94.0 ± 2.3	83.0 ± 7.9	74.3 ± 5.0	58.1 ± 15.4

42 per cent having escaped trouble with the authorities. The difference between the total prospect for Group IV and Group I at the age of 40 years is 28.4±6.9 per cent, and is therefore significant. If we calculate the chance of going through life

Table 65. **Average annual prospect and calculated total prospect in per cent for men, born in 1917 or later, of escaping action by the society because of serious crimes, inebriety, offences of drunkenness or vagrancy.**

Age	Group I	Group II	Group III	Group IV
Average annual prospect				
15—20	96.9 ± 2.8	89.4 ± 7.1	87.9 ± 3.9	85.7 ± 3.4
20—25	96.6 ± 4.3	89.7 ± 10.7	90.8 ± 4.5	94.0 ± 2.6
Calculated total prospect				
20	85.5 ± 6.2	57.0 ± 15.8	52.5 ± 8.8	46.2 ± 7.7
25	72.0 ± 10.3	33.1 ± 20.9	32.4 ± 10.3	33.9 ± 8.1

Table 66. **Average annual prospect and calculated total prospect for men, born in 1917 or later, of escaping action by the society because of crimes entered in the Penal Register, inebriety or offences of drunkenness, vagrancy, or inadequate ability to support themselves.**

Age	Group I	Groups II + III	Group IV
Average annual prospect			
15—20	97.0 ± 3.0	85.4 ± 4.0	84.1 ± 3.6
20—25	95.3 ± 5.1	85.4 ± 5.7	90.5 ± 3.3
Calculated total prospect			
20	85.9 ± 6.6	45.5 ± 9.0	42.1 ± 8.0
25	67.6 ± 11.9	20.7 ± 10.7	25.5 ± 8.6

without being subject to intervention of the abovementioned kinds but also take offences of drunkenness into account, we find still worse prospects (Table 65). At 25 years of age the men in Group I are blameless in these respects in about 70 per cent (as against 80 per cent in Table 63). In the three other groups the chance has decreased to about half or from 50—60 per cent to about 32 per cent. (Cf. Table 63.) There is no greater difference between Groups II, III and IV.

It may be of interest to investigate which number of men is blameless if regard is also paid to inadequate ability of self-support, even if this circumstance cannot be considered as a

social failure of the same significance as the other kinds of misbehaviour. We find that also now the prospects are best for Group I (68 per cent) and worse for the other groups. In Groups II—IV only about 20—25 per cent of the individuals are blameless at 25 years of age (Table 66).

If the sons' percentual distribution into social classes is compared with that of the parents (the fathers), we find that Group I shows a perceptible difference. In this group the sons have, on an average, reached a better social position than the one occupied by the parents at the time of the child's detention. The same tendency can be seen in Group II. In both these groups the percentage of sons in social class II is somewhat higher than that of the parents. Groups I and II together yield 13.4 ± 2.4 per cent sons in class II; the corresponding figure for the parents is only 5.3 ± 1.6 per cent. The difference is 8.1 ± 2.9 per cent and may be considered probable. (Table 54, p. 250.) There is no such tendency in Groups III and IV. It should be pointed out that not all the data — and this applies particularly to those of older date — as to occupation and social status can be unduly trusted, though they do suggest that the sons in Groups I and II have shown themselves more adequately equipped to better themselves socially than the sons in the other groups.

The distribution into social classes does not vary very much from group to group, however, though if a comparison is made with the adult male population in Malmö and other towns in the county of Malmöhus (according to election statistics), we find the boys in our groups appreciably over-represented in the lowest class.

The risk figures for the *women* are low in all groups. Group I has a better prognosis in respect of future criminality, while the few girls in Group IV have the worst. The difference between Groups I and III as regards the total risk at 30 years of age is 15.9 ± 4.5 per cent, and between Groups I and IV 32.1 ± 13.4 per cent; in the former case it is significant. See Fig. 12 (p. 278).

Group I seems to show more favourable figures only as regards vagrancy. In any case the material is far too small to warrant definitive conclusions. See Fig. 15 (p. 280).

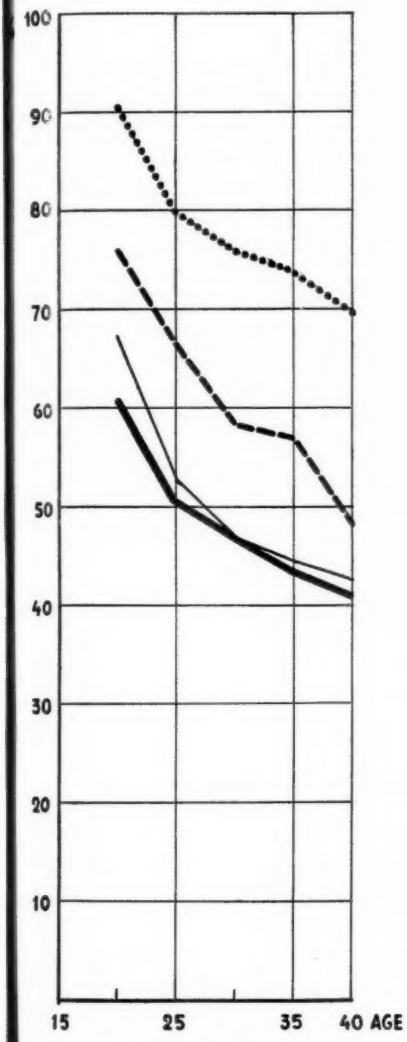


Fig. 18: Men.

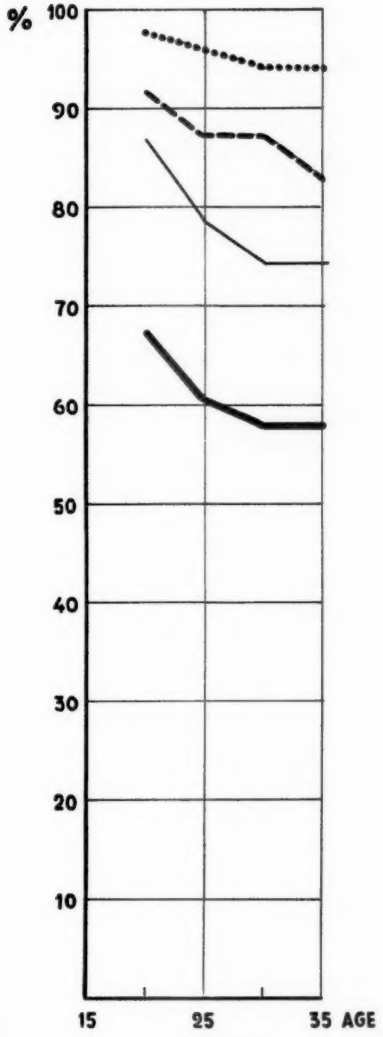


Fig. 19: Women.

Figs. 18 and 19. Calculated total prospect in per cent of escaping intervention by the society because of serious crimes, inebriety or vagrancy for men and women born in 1900 or later in groups I—IV.
Group I: dotted line, group II: broken line, group III: whole fine line, group IV: whole thick line.

Group I also shows better figures for the receipt of public assistance than the other groups; see Fig. 17 (p. 281). The difference as regards the total risk of assistance at 40 between Groups I and III is 24.1 ± 6.1 per cent, and is significant.

If we calculate the different prospects of managing without intervention of the previously mentioned kinds (with the exception of public assistance), we find that Group I comes off best here, too; see Table 64, p. 282 and Fig. 19. If the few girls in Group IV are left out of account, and Group I is compared with Group III, we get a significant difference of 19.7 ± 5.5 per cent for the total risk at the age of 35.

It is difficult to compare the social status in the different groups, due partly to the smallness of the material, partly to the fact that the status of the married women is determined by the occupation of their husbands. There is no doubt that more of them land up in the lowest class than is the case in the total population. In this respect, the distribution agrees well with that of the boys.

It is clear, then, that for both sexes Group I has stood out from the others and proves to be the one with the best development. We shall now discuss how this different development is to be explained.

To recapitulate: the children in Group I all come from bad homes, and have all been taken in charge for prophylactic ends. Their first and original environment has often been as bad as it could be (see Chap. VI). In addition, the hereditary taint was worst here, as regards both criminality and abuse of alcohol (see Chap. VII). Nor were they removed to other surroundings any too early — on an average, not until they had reached school age (see Chap. IV).

There is therefore every reason to expect a considerably less favourable development for this group, and it is remarkable that its members have nevertheless managed best as adults. This may be explained, at least in part, by the different principles of selection. The children in Group I have been selected not as individuals but on account of the badness of their homes, and showed no signs of delinquency or maladjustment when taken in charge.

The children in Group II are also from unfavourable environments, but they already show individual features of the delinquency type which, even if not severe enough to necessitate the child's removal from the home, must nevertheless colour its subsequent development. In Groups III and IV, the selection has been dictated exclusively by the personal disposition and behaviour of the individuals.

It therefore seems as if the individual disposition has had most bearing on the subsequent development, and that the original environment has not been so important. We have evidence of this in the fact that a number of persons both in Group III and, above all, in Group IV, were from good homes, though this did not stop them either from showing symptoms of early maladjustment to society in different ways, or from turning criminal later on.

CHAPTER XV.

PROGNOSIS IN REGARD TO CERTAIN SPECIAL CHARACTERS

INTRODUCTION.

In the previous chapter a general prognosis has been given for various groups of children, namely those coming from bad homes without themselves showing any symptoms of delinquency (Group I), those who both came from bad homes and showed some degree of delinquency, who thus form a mixed group (Group II), a third group who were definitely delinquent (Group III) and finally a group of older children who had been sentenced for crime.

It is however of interest to note to what extent the prognosis is different, not only for these groups, but also for children having special qualities. A comparison is made between legitimate and illegitimate children, unusually stupid children with those of more normal intelligence, and finally children from specially bad homes with those from good homes. In this connection the prognosis for children of parents with a special handicap in respect to a tendency to criminality etc. is also investigated.

As in previous investigations we are concerned with the extent to which children in these different categories become criminal, i.e. are entered in the Penal Register; the extent to which they become alcoholics, i.e. are reported to the Temperance Board or are fined for drunkenness; the extent to which they are taken up for vagrancy; to what extent they become unable to support themselves, or, more correctly, the extent to which they receive

public assistance. In the following pages we shall discuss the special problems of prognosis with reference to the factors here mentioned.

PROGNOSIS AND ILLEGITIMACY.

We have already investigated the extent to which illegitimate children are taken in charge by the Child-Welfare Board. Even though this occurs to a specially great extent among them, this does not necessarily mean that the prognosis for such children is specially bad. Of course their upbringing is more haphazard than that of other children as the mother cannot look after them, but in their later development they may turn out as well as other children.

In order to find out how matters stood in this respect all the cases of illegitimacy in each of the four groups were collected. These children were compared with a series of legitimate children consisting of proportionate numbers of children from each group. This was done in order to produce a proportionately weighted series affecting the risk and thus to make the figures comparable.

It may be noted that illegitimate children were in a minority in all the groups. The making of a weighted series thus meant that the number of legitimate children was diminished to equal the number of illegitimate ones.

1. Illegitimacy and criminality.

If we first take a look at the figures for risk in Table 67 (a), we find that the annual risk appears to be greater for illegitimate boys in the younger age-groups than for legitimate ones. The difference for the age-group 15—20 years is 2.4 ± 1.8 per cent and thus is not significant. After the age of 25 years however the risk of becoming a criminal appears to be less for the illegitimate. A comparison of the total risk of criminality for all ages up to 40 therefore shows no definite difference between the two categories. This good correspondence however does not exclude the possibility of variations among the different groups.

Table 67. **Legitimate and illegitimate birth and prognosis (men). Average annual risk and calculated total risk in per cent for men of legitimate and illegitimate birth and of different ages. Groups I—IV together.**

Average annual risk per cent			Calculated total risk per cent		
Age, years	Illegitimate birth	Legitimate birth (Weighted percentages)	Age, years	Illegitimate birth	Legitimate birth (Weighted percentages)
	a. *Birth in relation to criminality*				
15—20...	6.1 ± 1.6	3.7 ± 0.8	20.....	27.0 ± 3.6	17.2 ± 1.7
20—25...	2.9 ± 1.2	2.4 ± 0.7	25.....	37.0 ± 4.1	26.6 ± 2.1
25—30...	0.7 ± 0.7	1.3 ± 0.6	30.....	39.0 ± 4.2	31.2 ± 2.3
30—35...	0.4 ± 0.6	0.6 ± 0.4	35.....	40.0 ± 4.3	33.2 ± 2.4
35—40...	0.3 ± 0.7	0.8 ± 0.6	40.....	41.0 ± 4.4	35.7 ± 2.5
40—45...	—	0.7 ± 0.8	45.....	41.0 ± 4.5	37.8 ± 2.8
	b. *Birth in relation to inebriety*				
15—20...	0.9 ± 0.7	0.4 ± 0.4	20.....	4.5 ± 1.5	2.2 ± 0.9
20—25...	1.5 ± 0.9	0.9 ± 0.3	25.....	11.4 ± 2.3	6.6 ± 1.2
25—30...	0.5 ± 0.6	1.1 ± 0.4	30.....	13.4 ± 2.6	11.6 ± 1.5
30—35...	0.8 ± 0.9	0.9 ± 0.5	35.....	16.9 ± 3.2	15.6 ± 1.7
35—40...	0.7 ± 1.1	1.3 ± 0.7	40.....	19.9 ± 3.8	21.0 ± 2.1
40—45...	—	1.5 ± 1.5	45.....	19.9 ± 6.4	26.9 ± 3.4
	c. *Birth in relation to vagrancy*				
15—20...	0.4 ± 0.4	0.4 ± 0.2	20.....	2.0	2.1
20—25...	1.0 ± 0.7	0.5 ± 0.2	25.....	7.1	4.4
25—30...	—	0.2 ± 0.2	30.....	7.1	5.3
30—35...	0.2 ± 0.3	0.1 ± 0.1	35.....	7.8	5.8
35—40...	—	0.03 ± 0.1	40.....	7.8	5.9
40—45...	—	—	45.....	7.8	5.9
	d. *Birth in relation to poor relief*				
15—20...	1.1 ± 0.7	1.2 ± 0.4	20.....	5.5	5.9
20—25...	2.9 ± 1.1	2.7 ± 0.5	25.....	18.6	18.0
25—30...	3.6 ± 1.4	2.8 ± 0.6	30.....	32.1	28.9
30—35...	2.7 ± 1.5	2.6 ± 0.7	35.....	40.9	37.7
35—40...	3.1 ± 2.1	2.9 ± 0.9	40.....	49.7	46.3
40—45...	1.2 ± 1.9	1.9 ± 1.1	45.....	52.7	51.2

Investigation of the various groups shows that in Group I the total risks are about the same for both categories, i.e. for men up to 40 years 23.4 per cent for the illegitimate and 23.7 per cent for the legitimate.

In Group II the position is about the same. For men up to 35 years old the risk is 36.9 per cent for the illegitimate and 33.8 per cent for the legitimate.

In Group III however it is remarkable that the risk is higher for the legitimate than for the illegitimate. Up to the age of 40 years the latter have a total risk av 39.9 per cent while the legitimate have an even higher risk, i.e. 48.8 per cent. It is unexpected to find a higher figure for the legitimate, but the result may be due to random variation.

Investigation of Group IV on the other hand, seems to show a higher risk for the illegitimate than for the rest. The difference in the annual risk is most marked between the ages of 15 and 20 years, where it amounts to 10.7 ± 5.2 per cent but even here it is not statistically significant. There is also a difference in the total risk up to the age of 30 years. The difference is here 37.3 ± 14.8 per cent and is thus statistically probable.

In general it may be said that as far as those who were taken in charge by the Child-Welfare Board as children are concerned, no difference in criminal tendency can be shown between the legitimate and the illegitimate. However in Group IV which consists of young criminals, it appears as though the prognosis were worse in this respect for the illegitimate than for the legitimate. Group IV by its very nature, consists of cases which have to some extent been selected, as only some of those sentenced were reported to the Child-Welfare Board. It is impossible to estimate the effect of this selection. It is therefore difficult to draw any conclusions from the figures given.

2. Illegitimacy and inebriety.

The next question which arises is whether there is any difference between the legitimate and the illegitimate in the matter of the abuse of alcohol in later life. Cf. Table 67 (b).

It must be pointed out that these figures only relate to those born since 1900 as Temperance Boards have only been in existence since 1916. It was thus impossible for those born before 1900 to be reported to the Temperance Board within the first year after their sixteenth birthday. It was therefore ne-

cessary to limit the material as described above. In the younger age-groups there is a small difference in the risk of becoming the object of measures by the Temperance Board. The figure is higher for the illegitimate, but it is insignificant both for the annual and the total risks, and is counterbalanced in the older age-groups so that it is not possible to establish any statistical difference for the material as a whole. If the different groups are examined separately it is found that those coming under the care of the Child-Welfare Board show no difference in this respect either. However, when we come to Group IV (young criminals) the position appears to be different, in that the total risk up to the age of 35 years appears to be nearly half as great again for the illegitimate as for the legitimate. The difference, 14.0 ± 10.7 per cent, however, may be due to random variation.

3 and 4. Illegitimacy and vagrancy and poor relief.

The investigations into both of these questions (risk of receiving a warning against vagrancy and risk of requiring some form of public assistance) gave negative results. No difference could be demonstrated between the legitimate and the illegitimate, either in the material as a whole, or in the different groups. Cf. Table 67 (c and d).

In general it can be said that no difference in prognosis can be demonstrated between men of legitimate and illegitimate birth who were taken in charge by the Child-Welfare Board during childhood. (Groups I, II and III.) Group IV (young criminals) appears to be exceptional in certain respects, specially where the risk of criminality in later life is concerned, but also in the matter of inebriety. However Group IV does not include all those of the appropriate age who were sentenced during the period since, as has previously been mentioned, those individuals who had previously been taken in charge by the Child-Welfare Board and who then committed crimes at this age are not included in this group. Furthermore it does not include cases belonging to the later part of the period who were sentenced but were then taken in charge by the Child-

Welfare Board. Again one cannot be absolutely sure that all cases of offences by young persons have been referred to the Child-Welfare Board for a report. The material is thus incomplete, for which reason it is hard to give any opinion as to the implications of the figures.

The only investigation carried out among the *women* was one concerning the relationship between legitimacy or illegitimacy and *vagrancy*. The figures for risk in the other categories are so low for women that no result is to be hoped for by collecting them. It appears that the illegitimate did not have a worse prognosis in respect of vagrancy than the legitimate, in that, at the age of 25 years the difference between the total risk for both categories is 5.2 ± 2.12 per cent and thus not even statistically probable. By the age of 35 years the difference is 3.9 ± 2.9 per cent and thus not probable. This suggests that the previous difference was not a real one. Cf. Table 68.

Table 68. **Legitimate and illegitimate birth and prognosis (women). Average annual risk and calculated total risk in per cent for women of legitimate and illegitimate birth of being warned or sentenced to compulsory labour because of vagrancy. Groups I—IV together.**

Average annual risk per cent			Calculated total risk per cent		
Age, years	Illegitimate birth	Legitimate birth (Weighted percentages)	Age, years	Illegitimate birth	Legitimate birth (Weighted percentages)
15—20	0.65 ± 0.72	0.42 ± 0.39	20	3.2 ± 1.6	2.1 ± 0.9
20—25	0.27 ± 0.43	1.61 ± 0.23	25	4.5 ± 1.9	9.7 ± 1.0
25—30	0.38 ± 0.60	0.35 ± 0.39	30	6.3 ± 2.3	11.3 ± 1.3
30—35	0.27 ± 0.60	0.05 ± 0.17	35	7.6 ± 2.6	11.5 ± 1.3
35—40	—	—	40	7.6 ± 2.7	11.5 ± 1.4

PROGNOSIS AND HOME CONDITIONS.

In investigations of this kind great importance has always been attached to the influence of the environment in which the child grew up. It is however true that even if it is discovered

how great a percentage of delinquent children come from bad homes, the significance of environmental factors in the origin and development of delinquency is not thereby established. As has previously been pointed out, bad hereditary qualities in the parents may lead to a bad prognosis; they give rise to a bad environment for the child as well as to a bad prognosis.

An analysis of the home conditions of the delinquent children has been made here also. It shows that the majority of cases in Groups III and IV came from bad or rather bad homes, but that some of these children came from homes which were described as good.

It is therefore of interest to see whether any relationship can be traced between the environment in which the child grew up and his later prognosis. Groups I and II where all children came from bad homes, have been left out of this analysis. As in the previous investigation concerning legitimacy, the children from bad homes in Groups III and IV have been combined and compared with children from good homes after proportionately weighted percentages of the latter had been derived from the different groups.

1. Home conditions and criminality.

Table 69 (a) shows that men coming from bad homes run a rather greater risk of committing crimes than those coming from good homes. In so far as the total risks for different ages are concerned we find differences here which are not only dependent on chance. The difference in total risk for the ages up to 40 years is 14.8 ± 5.9 per cent and thus statistically probable. If Groups III and IV are separately investigated in this respect a similar result is found though it is not quite as obvious in Group III. In Group IV, however, it is more conspicuous and the risk of committing a second crime by the age of 40 is twice as great for those coming from bad homes as for those from good ones. The difference is 25.4 ± 8.9 per cent and thus statistically probable.

Table 69. **Character of the home and prognosis (men). Average annual risk and calculated total risk in per cent for men of different ages from good or bad homes. Groups III and IV together.**

Average annual risk per cent			Calculated total risk per cent		
Age, years	Bad homes	Good homes (Weighted percentages)	Age, years	Bad homes	Good homes (Weighted percentages)
a. *Character of the home in relation to criminality*					
15—20...	8.2 ± 1.4	5.3 ± 1.7	20.....	34.7 ± 3.1	23.9 ± 3.9
20—25...	3.6 ± 0.9	1.9 ± 1.0	25.....	45.7 ± 3.4	30.8 ± 4.2
25—30...	1.4 ± 0.7	1.1 ± 0.9	30.....	49.3 ± 3.5	34.5 ± 4.4
30—35...	0.7 ± 0.6	0.2 ± 0.4	35.....	51.0 ± 3.5	35.0 ± 4.5
35—40...	0.4 ± 0.5	0.7 ± 0.9	40.....	52.0 ± 3.6	37.2 ± 4.7
40—45...	0.5 ± 0.8	—	45.....	53.0 ± 3.7	37.2 ± 4.7
b. *Character of the home in relation to inebriety*					
15—20...	0.7 ± 0.4	1.0 ± 0.8	20.....	3.3 ± 0.9	4.8 ± 1.8
20—25...	1.7 ± 0.6	0.6 ± 0.6	25.....	11.0 ± 1.6	7.6 ± 2.2
25—30...	1.5 ± 0.7	0.3 ± 0.4	30.....	17.5 ± 2.1	8.8 ± 2.4
30—35...	1.0 ± 0.7	0.4 ± 0.7	35.....	21.4 ± 2.4	10.8 ± 2.7
35—40...	1.4 ± 1.0	0.2 ± 0.6	40.....	26.8 ± 3.0	11.7 ± 2.9
40—45...	0.5 ± 1.2	1.5 ± 2.6	45.....	28.7 ± 3.6	18.1 ± 5. 9
c. *Character of the home in relation to vagrancy*					
15—20...	0.7 ± 0.4	0.3 ± 0.4	20.....	3.4 ± 0.9	1.3 ± 0.8
20—25...	0.6 ± 0.4	0.4 ± 0.4	25.....	6.5 ± 1.2	3.1 ± 1.2
25—30...	0.3 ± 0.1	0.1 ± 0.3	30.....	7.6 ± 1.2	3.8 ± 1.3
30—35...	0.1 ± 0.3	—	35.....	7.9 ± 1.4	3.8 ± 1.4
35—40...	0.2 ± 0.3	—	40.....	8.8 ± 1.5	3.8 ± 1.4
40—45...	—	—	45.....	8.8 ± 1.6	3.8 ± 1.5
d. *Character of the home in relation to poor relief*					
15—20...	1.3 ± 0.5	0.6 ± 0.6	20.....	6.3 ± 1.2	3.0 ± 1.3
20—25...	3.5 ± 0.8	1.9 ± 0.9	25.....	21.6 ± 2.2	11.8 ± 2.3
25—30...	4.1 ± 1.1	1.9 ± 1.1	30.....	36.4 ± 2.9	19.9 ± 3.1
20—35...	2.7 ± 1.1	2.2 ± 1.3	35.....	44.5 ± 3.3	28.3 ± 3.9
35—40...	3.2 ± 1.4	2.4 ± 1.6	40.....	56.4 ± 3.8	36.5 ± 4.7
40—45..	2.8 ± 1.8	1.6 ± 1.9	45.....	62.2 ± 4.2	41.5 ± 5.4

2. Home conditions and inebriety.

The same relationship can be demonstrated in respect of the risk of being the subject of measures by the Temperance Board,

i.e. men whose parental home was bad run a significantly greater risk in this respect than those coming from good homes. Cf. Table 69 (b). The difference in the total risk by the age of 35 years is statistically probable and by the age of 40 it is significant. The same conditions hold for each group treated separately but the difference is most pronounced in Group III where the total risk of becoming the subject of measures by the Temperance Board by the age of 40 years is greater for those coming from bad homes than for those who enjoyed the advantages of a good home. The difference by the age of 40 years is 21.2 ± 7.6 per cent and is statistically probable.

3. Home conditions and vagrancy.

The difference between those from bad and good homes in respect to the risk of receiving a warning against vagrancy is, as is seen in Table 69 (c), less. However it is present both in the figures for the annual risk and specially in the figures for the total risk where the differences by the ages of 25 years and 30 years are 3.4 ± 1.7 per cent and 3.8 ± 1.8 per cent respectively.

4. Home conditions and poor relief.

It may be thought that ability to be self-supporting, as reflected by the necessity of requesting public assistance, would have little relation to the original home-conditions of the person concerned. Nevertheless it is shown that even in this respect the nature of the parental home is not without influence in that children from good homes had considerably greater prospects of success in the economic struggle for existence than those coming from bad ones. The difference between the total risks by the age of 45 years is 20.7 ± 6.8 per cent and thus significant. If Groups III and IV are treated separately the same results are found, most markedly in Group IV. Cf. Table 69 (c).

To sum up, these results suggest that, in all the respects which have been investigated, the nature of the home was of decisive importance for the social prognosis. This is the case in respect of later criminality, of being the subject of measures by the Temperance Board, of vagrancy, and of incapacity for being self-supporting.

As in the previous investigation concerning legitimacy, the only investigation carried out on the *women* was into the relationship between home-conditions and *vagrancy*. Here there is a marked difference in that none of the girls from good homes abandoned themselves to vagrancy, all those who did came from homes of the worst character. The calculated total risk by the age of 25 years is 15.3 ± 3.7 per cent and by 35 years it is 20.6 ± 4.5 per cent. This is the case whether the groups are treated together or each group is treated by itself.

PROGNOSIS AND INTELLECTUAL LEVEL.

The information as to the intellectual capacity of the children included in this material is all derived at second hand and is thus to some extent subject to error. (Cf. Chapter IX). Even with this reservation it is worth while investigating at a later date whether those who seemed to be of normal intelligence were more successful than the others and above all whether it was the weak class and feeble-minded children who were the most important factor causing the bad prognosis.

1. Intellectual level and criminality.

No significant statistical difference can be demonstrated for the material as a whole between the categories described as »of weak intelligence», »feeble-minded» (debile) and »of normal intelligence». It is seen from Table 70 (a) that both the annual and the total risks are of much the same size for all three categories. It appears as though there were a rather higher risk of committing a crime among the feeble-minded but owing to the large standard errors no definite statement can be made.

Examination of the groups separately shows a certain tendency towards a worse prognosis for the feeble-minded but, as in the material as a whole, the difference is small; only in Group IV is it more pronounced. It is however possible that certain differences in distribution would have been found had a larger material exhibiting more pronounced differences in intelligence, been available.

Table 70. **Intellectual level and prognosis (men). Average annual risk and calculated total risk in per cent for men of different ages and of different intellectual level of committing a crime, being reported to the Temperance Board because of inebriety, being warned or sentenced for vagrancy, or of having to apply for poor relief. Groups I—IV together.**

Average annual risk per cent				Calculated total risk per cent			
Age, years	Weak intelligence (I. Q. 90—80)	Feeble-mindedness (I. Q. < 80)	Normal intelligence (Weighted percentages	Age, years	Weak intelligence (I. Q. 90—80)	Feeble-mindedness (I. Q. < 80)	Normal intelligence (Weighted percentages)
a. *Intellectual level in relation to criminality*							
15—20	6.3 ± 1.9	8.7 ± 3.4	6.4 ± 1.0	20	27.8	36.5 ± 7.5	28.1 ± 2.3
20—25	2.8 ± 1.3	4.3 ± 2.5	2.5 ± 0.6	25	37.4	49.0 ± 8.3	36.7 ± 2.5
25—30	0.8 ± 0.8	1.6 ± 2.1	1.1 ± 0.5	30	39.9	53.0 ± 8.6	40.2 ± 2.6
30—35	0.6 ± 0.8	2.6 ± 3.4	0.5 ± 0.4	35	41.9	58.8 ± 9.3	41.7 ± 2.7
35—40	1.0 ± 1.3	1.3 ± 2.9	0.6 ± 0.5	40	44.9	61.3 ± 9.7	43.2 ± 2.7
40—45	0.9 ± 2.0	.	0.6 ± 0.7	45	47.4	.	44.7 ± 2.9
b. *Intellectual level in relation to inebriety*							
15—20	0.7 ± 0.7	1.2 ± 1.3	0.8 ± 0.4	20	3.3 ± 1.5	5.9 ± 3.0	3.7 ± 0.8
20—25	1.8 ± 1.1	1.4 ± 1.4	1.2 ± 0.5	25	11.8 ± 2.7	12.2 ± 4.2	9.5 ± 1.3
25—30	1.1 ± 1.0	1.1 ± 1.7	0.8 ± 0.4	30	16.8 ± 3.3	16.9 ± 5.4	12.9 ± 1.6
30—35	0.4 ± 0.7	—	1.2 ± 0.6	35	18.5 ± 3.5	16.9 ± 6.7	18.1 ± 2.0
35—40	1.9 ± 1.9	—	0.9 ± 0.7	40	25.9 ± 4.9	16.9 ± 9.3	21.7 ± 2.4
40—45	.	.	.	45	.	.	.
c. *Intellectual level in relation to vagrancy*							
15—20	1.0 ± 0.9	0.3 ± 0.6	0.5 ± 0.3	20	5.1 ± 2.1	1.3 ± 1.3	2.4 ± 0.6
20—25	1.0 ± 0.7	1.2 ± 1.2	0.3 ± 0.2	25	9.8 ± 2.6	6.9 ± 2.9	3.7 ± 0.8
25—30	0.4 ± 0.5	0.4 ± 0.9	0.1 ± 0.1	30	11.6 ± 2.8	8.8 ± 3.5	4.0 ± 0.8
30—35	—	0.6 ± 1.4	0.1 ± 0.2	35	11.6 ± 2.8	11.6 ± 4.5	4.6 ± 0.9
35—40	—	—	—	40	11.6 ± 2.9	11.6 ± 4.8	4.6 ± 1.0
40—45	—	.	.	45	11.6 ± 2.9	.	4.6 ± 1.1
d. *Intellectual level in relation to poor relief*							
15—20	1.7 ± 1.0	1.8 ± 1.5	0.9 ± 0.4	20	8.2	8.7 ± 3.4	4.5 ± 0.8
20—25	2.8 ± 1.2	4.5 ± 2.2	2.4 ± 0.6	25	20.4	27.4 ± 4.6	15.4 ± 1.5
25—30	2.9 ± 1.4	3.8 ± 2.9	3.2 ± 0.8	30	31.3	40.3 ± 7.4	28.1 ± 2.1
30—35	2.4 ± 1.5	4.7 ± 4.3	2.5 ± 0.8	35	39.1	53.0 ± 9.3	36.7 ± 2.5
35—40	3.6 ± 2.5	.	2.6 ± 1.0	40	49.3	.	44.5 ± 2.9
40—45	4.3 ± 4.3	.	2.4 ± 1.4	45	59.3	.	50.8 ± 3.9

2. Intellectual level and inebriety.

No difference can be demonstrated in regard to risk of being subject of measures by the Temperance Board between the normal, weak class or feeble-minded individuals in the material as a whole. If the groups are examined separately it is found that in Groups I—III the number of feeble-minded is too small to permit the drawing of any conclusions. In Group IV no difference in total risk can be found for the different degrees of intelligence but the risk is about equally distributed among the three categories.

3. Intellectual level and vagrancy.

It was to be expected that there would be some correlation between poor intelligence and the risk of being the subject of measures against vagrancy. It is however hardly possible to demonstrate such a connection. There seems to be a difference between the normal and the feeble-minded but this is not statistically significant. Table 70 (c). It must be pointed out also that the material is too small to permit the drawing of any definite conclusions for the separate groups.

4. Intellectual level and poor relief.

It would also have been expected that the risk of requiring public assistance would have been greater among the feeble-minded (Table 70 (d)). The figures for the annual risk however are very varying for the weak class children, the feeble-minded and normal children. Owing to the large standard errors the figures are not reliable but a tendency may be observed for the figures for the group with normal intelligence to be more stable. The difference between the total risks for those of normal intelligence and the feeble-minded by the age of 35 years is 16.3 ± 9.6 per cent and thus is not even probable. The material is too small to draw any definite conclusions as to the conditions in the groups separately.

Thus it was not possible to demonstrate any prognostic difference associated with different degrees of intelligence within the material. It is possible that differences might have been

Table 71. **Intellectual level and prognosis (women). Average annual risk and calculated total risk in per cent for women of different intellectual level of being warned or sentenced to compulsory labour because of vagrancy, or of having to apply for poor relief. Groups I—IV together.**

Average annual risk per cent				Calculated total risk per cent			
Age, years	Weak intelligence (I. Q. 90—80)	Feeblemindedness (I. Q. V 80)	Normal intelligence (Weighted percentages)	Age, years	Weak intelligence (I. Q. 90—80)	Feeblemindedness (I. Q. V 80)	Normal intelligence (Weighted percentages)
a. *Intellectual level in relation to vagrancy*							
15—20	0.4 ± 0.8	0.7 ± 1.6	0.8 ± 0.6	20	1.9 ± 1.9	3.5 ± 3.6	3.9 ± 1.3
20—25	1.5 ± 1.5	2.2 ± 2.2	0.8 ± 0.6	25	9.1 ± 3.8	13.7 ± 5.9	7.4 ± 1.8
25—30	0.9 ± 1.4	0.9 ± 2.0	0.4 ± 0.5	30	13.2 ± 4.8	17.4 ± 7.0	9.2 ± 2.0
30—35	0.6 ± 1.3	·	—	35	15.7 ± 5.3	·	9.2 ± 2.1
35—40	1.0 ± 2.2	·	—	40	19.7 ± 6.8	·	9.2 ± 2.1
b. *Intellectual level in relation to poor relief*							
15—20	0.7 ± 1.1	1.4 ± 2.2	1.2 ± 0.7	20	3.5	6.8 ± 5.0	5.9 ± 1.7
20—25	2.1 ± 1.8	6.9 ± 3.8	2.0 ± 0.9	25	13.2	34.8 ± 7.9	14.9 ± 2.6
25—30	1.7 ± 1.9	2.9 ± 3.6	0.6 ± 0.6	30	20.4	43.7 ± 10.7	17.4 ± 2.8
30—35	1.2 ± 1.9	·	1.0 ± 0.9	35	25.0	·	21.4 ± 3.2
35—40	·	·	0.5 ± 0.8	40	·	·	23.4 ± 3.5

found had a larger material been used. It is also possible that the rather uncertain evaluation of the intelligence of the individuals in the material played a part in producing this result. On the other hand the question arises as to whether intellectual capacity in itself plays an important part in determining the occurrence of crime, delinquency and asocial behaviour in general, or whether other psychological qualities such as temperament etc. play so great a part that they completely dominate all other factors and thus in fact prevent an investigation of this kind from showing results. It is possible that the result might have been different if we had been able to divide the material up according to differences of temperament.

For the reason given above a comparison has not been made for *women* between the different categories of children and the different aspects of their prognosis except in relation to

vagrancy (Cf. Table 71) and in relation to the risk of requiring poor relief on grounds of destitution. The investigation of the relationship between vagrancy and different levels of intelligence is of special interest as it has often been stated, in investigations of women given to vagrancy, that most of them are of low intelligence (KEMP, KNÖS). In this material however, no definite difference can be shown, but it appears as though vagrancy were just as common among girls of normal intelligence as among rather backward or feeble-minded ones. However the material is small and the standard error large so that no definite statement can be made.

There is also a certain difference between women of normal intelligence and feeble-minded ones in respect of the risk of requiring *poor relief* on grounds of destitution. By the age of 30 years the difference in the total risk for the two groups is 26.3 ± 11.1 per cent which is not however even probable.

QUALITIES OF THE PARENTS AND PROGNOSIS OF THE CHILDREN.

Having thus tried to find some correlation between various aspects of the prognosis and certain environmental conditions such as good or bad homes, an attempt will now be made to demonstrate any relationship between these aspects of the prognosis and certain qualities in the parents. In doing so attention will only be paid to those factors about which we have definite and objective information, i.e. whether the parents were criminals or not and whether they were drunkards or not. An investigation will first be made in respect of criminality among the parents.

1. Criminality of the parents and the children.

It is natural to wonder whether criminal parents have criminal children to a greater extent than parents who are not criminals. Table 72 shows this probably to be the case.

The difference between the total risk for men with and without criminal parents appears at all ages and is statistically

Table 72. **Average annual risk and calculated total risk in per cent for individuals with or without criminal parents of being convicted of crimes recorded in the Penal Register. Groups I—IV together.**

Average annual risk per cent			Calculated total risk per cent		
Age, years	One of the parents convicted of crime	None of the parents convicted of crime (Weighted percentages)	Age, years	One of the parents convicted of crime	None of the parents convicted of crime (Weighted percentages)
			Men		
15—20...	7.6 ± 1.9	4.7 ± 0.7	20.....	32.6 ± 4.2	21.5 ± 1.7
20—25...	4.3 ± 1.6	2.1 ± 0.5	25.....	45.9 ± 4.8	29.3 ± 1.9
25—30...	1.2 ± 1.0	1.1 ± 0.4	30.....	48.9 ± 4.9	33.2 ± 2.0
30—35...	0.7 ± 0.9	0.5 ± 0.3	35.....	50.7 ± 5.0	34.7 ± 2.0
35—40...	0.4 ± 0.9	0.8 ± 0.5	40.....	51.7 ± 5.1	37.2 ± 2.1
40—45...	0.8 ± 1.8	0.4 ± 0.5	45.....	53.7 ± 5.5	38.3 ± 2.2
			Women		
15—20...	0.9 ± 1.0	0.8 ± 0.5	20.....	4.5	4.0
20—25...	1.8 ± 1.4	0.9 ± 0.5	25	12.8	8.2
25—30...	1.6 ± 1.6	0.4 ± 0.4	30.....	19.6	10.2
30—35...	—	0.2 ± 0.3	35.....	19.6	11.2
35—40...	—	—	40.....	19.6	11.2

probable for all ages and at 25 years it is statistically significant. The same tendency appears among the women, but here the material is small and no significant differences can be established. In going through the different ages the same difference is found in that women with criminal parents showed a higher risk throughout than those without.

2. Abuse of alcohol by the fathers and the prognosis of the children.

There remains the question as to whether there is any relation between the abuse of alcohol by fathers and the prognosis for their children. In order only to take into account cases as to which there was definite and objective evidence of drunkenness, only fathers who have been the subjects of measures by the Temperance Board are included. Mothers are not taken into account as the number of women who are drunkards is

relatively small; the mothers were therefore not even looked for in the register of the Temperance Board.

Prognosis as regards criminality.

If the situation is first examined in relation to the criminal activities of the *men* it is seen from Table 73 that the total risk of crime at all ages is higher for those whose fathers are described as inebriates than for the rest. The differences in the total risk for all the age-groups are statistically probable.

Table 73. **Average annual risk and calculated total risk in per cent of being sentenced for crimes recorded in the Penal Register for individuals whose fathers were inebriates or not. Groups I—IV together.**

Average annual risk per cent			Calculated total risk per cent		
Age, years	The father inebriate	The father not inebriate (Weighted percentages)	Age, years	The father inebriate	The father not inebriate (Weighted percentages)
		Men			
15—20	7.8 ± 2.5	5.3 ± 0.9	20	33.3 ± 5.5	23.9 ± 2.1
20—25	4.5 ± 2.0	1.4 ± 0.5	25	47.0 ± 6.3	29.0 ± 2.3
25—30	1.9 ± 1.7	0.8 ± 0.4	30	51.8 ± 6.6	31.9 ± 2.4
30—35	1.0 ± 1.6	0.4 ± 0.4	35	54.3 ± 6.8	33.4 ± 2.5
35—40	2.1 ± 3.4	0.8 ± 0.6	40	58.9 ± 7.7	35.9 ± 2.6
		Women			
15—20	1.7 ± 1.9	0.9 ± 0.6	20	8.2 ± 4.2	4.5 ± 1.3
20—25	2.7 ± 2.5	0.7 ± 0.5	25	19.9 ± 6.6	7.9 ± 1.6
25—30	0.8 ± 1.8	0.5 ± 0.5	30	22.9 ± 7.3	10.4 ± 1.9
30—35	.	0.1 ± 0.2	35	.	10.9 ± 2.0

The different groups within the material will now be examined separately from this point of view. In Group I, which consists of children taken in charge on account of the badness of their homes and where inebriety was prevalent of over half (58.5 per cent) in the homes of the boys, there is no great difference in this respect, either in the annual, or the total risk.

In Group II, where the homes are also, by definition, bad, and where inebriety prevailed of nearly half in the homes of the boys (46.3 per cent), there is found to be considerable

difference for the figures for risk between the men whose fathers were severely addicted to alcohol and thus became the subjects of measures by the Temperance Board and those whose fathers were not so addicted. The material is however too small for a definite conclusion to be drawn.

Group III consists of true cases of delinquency. A fair proportion of these came from good homes but in about 23 per cent of the cases the father was addicted to drunkenness. Here also it is seen that both the annual and the total risk of committing a crime are higher for men at all ages if their fathers are described as drunkards than if they are not.

Group IV, the young criminals, shows the same state of affairs as the previous groups, perhaps even more markedly. The total risk for the sons, of committing crimes at the ages of 20 and 25 years is $^1/_3$ as high again for those whose fathers were confirmed inebriates as for those whose fathers were not.

In summing up it appears that whether the whole material or the different groups are examined separately the same conditions are found, namely that the sons run a considerably greater risk of being convicted of a serious crime if the fathers were addicted to alcohol to such an extent as to be subject to intervention by the Temperance Board.

Investigation of the risk of criminality among *women* whose fathers were inebriates gives no definite results; the material is however too small.

Prognosis as regards inebriety.

It may be asked whether the children of drunkards themselves become addicted to alcohol when they grow up.

An investigation was made of the material as a whole. It was made in the usual way using weighted percentages. It showed that the sons of fathers described as drunkards became addicted to drunkenness to a greater extent than those whose fathers did not abuse alcohol. Calculation of the total risk shows that at the age of 35 the difference between the two categories is almost statistically probable. (Table 74.)

Table 74. **Average annual risk and calculated total risk in per cent for men whose fathers were subjected to measure by the Temperance Board, of being themselves reported to the Temperance Board because of inebriety. Groups I—IV together.**

Average annual risk per cent			Calculated total risk per cent		
Age, years	The father inebriate	The father not inebriate (Weighted percentages)	Age, years	The father inebriate	The father not inebriate (Weighted percentages)
15—20	0.5 ± 0.6	0.7 ± 0.3	20	2.3 ± 1.3	3.4 ± 0.7
20—25	2.1 ± 1.3	1.2 ± 0.4	25	12.2 ± 3.1	9.0 ± 1.1
25—30	2.7 ± 1.9	0.9 ± 0.4	30	23.3 ± 4.8	12.8 ± 1.4
30—35	2.1 + 2.1	0.7 ± 0.4	35	31.2 ± 6.0	15.8 ± 1.6
35—40	1.8 ± 2.9	1.0 ± 0.6	40	37.2 ± 7.4	20.0 ± 2.0

Examination of the different groups shows the difference to be least marked in Group II.

It is thus seen that it is considerably more usual for boys whose fathers are criminals or drunkards themselves to be criminals or drunkards as the case may be, than for those whose fathers are not in these categories.

CRIMINALITY AND LENGTH OF DETENTION.

As well as these relationships in various respects, it would be of practical value to investigate whether the measures taken by society, i.e. removal from the home environment, showed any relationship between the later prognosis and, for example, the length of detention or the age of the individual at the time of the intervention.

No relationship can be found within the different groups between long or short periods of detention and the later prognosis. In Group I, children coming from a specially bad environment, it might have been expected that the longer the period of detention, and therefore the longer the children stayed away from the bad home-environment, the better the result would have been. This cannot however be shown.

Neither can any relationship be found between the length of the period of detention and the later prognosis in Groups II and III (slighter and more severe cases of delinquency). It may be supposed that children who proved difficult to bring up, and therefore were taken in charge for a long time, would show the same qualities later, which might have the effect of giving a worse prognosis to those with a longer period of detention, but it cannot be demonstrated in this material.

CRIMINALITY IN RELATION TO THE AGE AT WHICH THE CHILD WAS TAKEN IN CHARGE.

Group I has been divided into three age-groups, under 7 years, 7—11 years and 11 years and over. If the total risk of committing a first crime is calculated for ages up to 40 years, it is found that there is a certain difference in favour of those who were taken in charge after the age of 7 years (Table 75). Similarly if a division is made into 2 groups, over and under 11 years, the prognosis is worst for those who were taken in charge earliest. This is thus the opposite of what was expected. The difference is however small, but it is somewhat more marked among men than among women. When thus divided the groups are rather small, so the standard error would be rather large. It is surprising that no definite difference can be demonstrated. The home-environment in the earliest years cannot be without importance for the future behaviour of the child. The explanation of the impossibility of showing any great difference may be partly found in the fact of the smallness of the material.

The significance of the factor of age cannot be viewed from the same standpoint where the other groups are concerned as regarding Group I since the individuals are taken in charge on account of their own activities. They can scarcely be taken in charge before the age of 7 years as, before this age one can hardly talk of delinquency in its true meaning.

It would be of great interest to see whether children who showed a tendency to misbehaviour at an early age had a

Table 75. **Calculated total risk in per cent of being sentenced for crimes recorded in the Penal Register in relation to age when taken in charge for individuals in Group I.**

Age, years	Men			Women		
	Age when taken in charge					
	< 7 years	7—11 years	≥ 11 years	< 7 years	7—11 years	≥ 11 years
20	10.6	7.7	7.3	3.0	3.0	—
25	18.4	16.5	13.6	6.5	5.0	—
30	20.4	18.0	16.1	8.5	7.0	—
35	20.4	19.5	16.1	8.5	7.0	—
40	(27.0)	25.0	20.1	8.5	7.0	—

Table 76. **Calculated total risk in per cent of being sentenced for crimes recorded in the Penal Register for boys under or over 11 years of age at the first contact with the Child-Welfare Board or the Police.**

Age, years	Group II		Group III	
	Age at the first contact with the Child-Welfare Board or the Police			
	< 11 years	≥ 11 years	< 11 years	≥ 11 years
20.....	24.6	18.9	19.9	28.6
25.....	27.1	32.9	31.9	39.9
30.....	30.1	39.7	37.2	43.8
35.....	31.6	39.7	40.0	46.3
40.....	31.6	(43.7)	43.2	46.8

worse prognosis than those who showed these manifestations for the first time at a later one. No such information can be found in this material. However it gives fairly definite information as to the age at which those concerned first came into contact with the authorities on account of misbehaviour (criminality). An investigation should therefore be made into how the prognosis for men was affected by whether this contact occurred early or late.

For this purpose the material has been divided into two age-groups, i.e. contact before or after 11 years of age, and the risks were calculated for both age-groups. (Table 76.) In Group II

where the children were taken in charge on account of mild symptoms of delinquency, but where the homes were also markedly bad, the annual risk for men aged between 15 and 20 years is higher for those who came into contact with the authorities before the age of 11 years than for those who came into contact later. The difference may however be due to random variation and is cancelled in the next age group. If the total risks by the age of 35 years are compared it is found, that the risk is higher for those who came into contact with the authorities later. The same applies to the women in the material though they are too few for any conclusions to be drawn. If the risk in this respect is examined for men in Group III who were taken in charge as children on account of more serious delinquency, the same conditions are found. The total risk is higher for those who manifested delinquency of such a nature that the authorities interfered, at a later age.

In Group I this investigation has only been made in respect of whether symptoms of delinquency occurred before the age of 15 years or not. (Table 77.) In this group there were no signs of delinquency at the time of intervention by the Board. In Group IV the same division was made, i.e. according to whether symptoms of delinquency were or were not present. The number of children who had previously shown symptoms of delinquency was too small for any further subdivision.

In Group I it is seen that for the 20-year-olds the total risk of committing a crime is 3 times as great for those who had previously been delinquent as for those who had not previously shown symptoms of delinquency. By the age of 40 years the total risk for men who had been delinquent before the age of 15 years is 3 times as great as for those who showed no symptoms of delinquency as children.

In Group IV it appears similarly that those children who were delinquent before the age of 15 years have the worst prognosis. (Table 77.) For men, after a period of 20 years of observation following their first crimes, the risk of recidivism is nearly $^1/_3$ as high against among those who belong to the earlier age-group in respect of their first delinquency. The prog-

Table 77. **Calculated total risk in per cent of being sentenced for crimes recorded in the Penal Register for boys under or over 15 years of age at the first sign of delinquency or at the first contact with the Child-Welfare Board or the Police.**

Group I			Group IV		
Age, years	Before 15 years of age		Number of years of observation after discharge	Age at the first contact with the Child-Welfare Board or the Police	
	Sign of delinquency	No sign of delinquency		< 15 years	≥ 15 years
20	16.0	5.5	5	35.5	25.8
25	32.2	9.6	10	38.5	30.8
30	37.2	10.6	15	42.5	33.8
35	37.2	12.1	20	43.5	34.8
40	(50.9)	13.7	25	46.9	37.0

nosis is, in other words, worse for those who showed symptoms of delinquency before they committed the crime for which they were sentenced, than for those who had never previously come into contact with the authorities on account of delinquency. This is, in fact, neither surprising nor new, but is merely a confirmation of that which has previously been demonstrated by DAHLBERG and LINDBERG (1944), namely that the outlook for the future is worst among prisoners who have a previous record of asocial behaviour.

In regard to the significance of factors concerning age it may thus be said: Concerning Group I (children from bad homes who were taken in charge for prophylactic reasons) it cannot be shown in this material, that the fact of having been taken in charge before or after 7 years of age has any significance for the prognosis in respect of criminality (Table 75).

Concerning Groups II and III (children taken in charge on account of slight or severe delinquency respectively), the total risk is higher for those who came into contact with the authorities at a later age than for those who were in this situation at an earlier age. Thus there is no indication that delinquency arising at an early age gives a worse prognosis. However, our investigations in this respect show plainly for both Group I and

Group IV, that symptoms of delinquency of any sort are a serious omen which foreshadow the future development (Tables 76 and 77). On the other hand no information is given by this material as to whether the fact of such symptoms first arising at an early or a late age gives any indication as to the final prognosis. It is quite possible that a larger material showing more pronounced age-differences would have given results in one direction or the other.

SUMMARY.

The character of and statistical prognosis for delinquents depends partly on how the cases are selected and partly on the character of the society in which they live. Therefore, as is seen from the review of the literature, different results are obtained in different countries and at different times. The literature has been reviewed very extensively with the object of showing that very detailed information is required before an opinion can be formed on any investigation. The present investigation concerns 1727 boys and 619 girls who were in contact with the Child-Welfare Board in Malmö between 1903 and 1940. The cases were followed up until January 1st, 1944.

The cases were divided into 4 groups, i.e. Group I, children who were taken in charge solely on account of bad home conditions; Group II, children who were taken in charge because their home-conditions were bad and also because they had committed some slight act of delinquency; Group III, definite cases of delinquency; Group IV, young persons between the ages of 15 and 21 years who were convicted of crimes and concerning whom the Child-Welfare Board submitted a report to the court before their conviction. These last, however, comprise only a part of the young persons who were convicted of crime during the period in question.

I. THE CASES WERE FIRST CHARACTERIZED.

1) In the first place it was established that delinquents comprised about 2 per cent of the boys and about 1 per cent of the girls in the corresponding age groups.

2) The average *age* before 1926 at the time of being taken in charge was found to be 7.8 years for boys and 8.7 years for girls in Group I (children from bad homes); 10.9 years for boys

and 11.5 years for girls in Group II (children taken in charge on a mixed indication); and 12.3 years for boys and 12.8 years for girls in Group III (definite cases of delinquency). In Group IV the average age of boys and girls was 16.4 and 16.5 years. After 1926 the age was as a rule higher depending on new laws. This was most marked for the girls in Group III where the increase of the mean amounted to 3.4 years. For the boys in the same group the increase was 1.9 years. For Groups II and IV the increase was about $^1/_2$—1 year. Only in Group I the mean age was lower during the later period; for boys about $^1/_2$ and for girls 2 years. It is only natural that the age is lowest for children belonging to Group I and highest for young persons belonging to Group IV. The *measures* taken were as follows. Children in Group I were, as a rule, placed in private homes. The majority of cases in Group II were also treated in this way, i.e. $^3/_4$ of the boys and $^1/_2$ of the girls were so placed before 1926. Since 1926 about $^1/_3$ of the boys as well as of the girls were placed in private homes, $^1/_3$ were placed in children's homes and $^1/_3$ were transferred to detention homes. In Group III placement in detention homes was the rule, but a fair number were placed in private homes. In Group IV the activities of the Board were only confined to the submission of a report.

3) The causes for which the children were taken in charge have been grouped into 4 different *types* of misdemeanour, i.e. offences against property, vagrancy, sexual offences and general delinquency. Combinations of these causes can naturally also occur.

Among the slighter cases of delinquency in boys (Group II) the commonest types are vagrancy which occurs in 60 per cent of the cases and offences against property which occur in 53 per cent of the cases. General delinquency occurs in rather less than $^1/_3$ of the cases, i.e. 29 per cent. Amongst boys committing serious delinquencies (Group III), offences against property occur in the majority of cases, i.e. 80 per cent, while 52 per cent were guilty of vagabondage and 15 per cent of general delinquency.

Among girls belonging to Group II, vagrancy is most common after which come offences against property and thirdly

general delinquency. Sexual offences occur to a rather small extent, but much more frequently during the later period when the age at which the children could be taken in charge was raised. In the earlier period when the limiting age was 15 years there was no conspicuous difference between boys and girls.

Also for girls who have shown delinquency of a more serious kind (Group III) vagrancy is most common and occurs in 53 per cent. In the second place come sexual offences and property crimes with about the same percentages, i.e. 44, respectively 43 per cent. Then follows general delinquency in 14 per cent. Comparing the figures for the two periods we find that a striking change has occurred in the frequency of property crimes and sexual offences. The property crimes decreased during the years 1926—1940 to about half of the percentage of the earlier period, whereas the sexual offences increased to about three times their previous percentage, i.e. the figure for 1903—25.

II. IN THE SECOND PLACE THE ENVIRONMENT FROM WHICH THE CHILDREN CAME WAS CHARACTERIZED.

4) *Illegitimacy* was more prevalent than in the general population. This is specially the case in Group I, children who stand in danger of becoming delinquent. About $^1/_3$ of these children are illegitimate. The figure is nearly as high in Group II but in Group III, the really delinquent children, the illegitimacy rate is not higher among the boys than in the rest of the population. It is, however, found to be definitely increased among the girls. Curiously enough, the figure in Group IV is lower than that for the general population.

5) Nearly half the children came from *broken homes*. In the earlier part of the period the commonest cause for this was that one of the parents was dead, but in the later part divorce was an equally common cause. The delinquent girls came from broken homes to a greater extent than did the delinquent boys. It was also commoner for the girls to have experienced a change of home during the years of childhood than for the boys.

6) Where the general character of the *home* is concerned it is of course the case that the children in Groups I and II all came from bad homes. About half of the children who showed definite delinquency came also from bad homes; this was more common among the girls than among the boys. Conversely, a greater number of the boys than of the girls came from homes which were described as good. On the whole the homes were poor and the parents belonged for the most part to the lowest classes of society. The girls, to a greater extent than the boys, come from poor homes and from the lowest classes of society. In general it appears that when they become delinquent, the girls have been exposed to a worse environment than the boys.

7) Systematic information has been collected from the Central Penal Register as to the criminal record of the *parents,* and from the records of the Temperance Board as to inebriety. It is shown that every fifth child had a criminal father, i.e. twice the rate for the general population. The figure for Group I (children from bad homes) is specially high. It amounts to 58.5 per cent for boys and 50.8 per cent for girls during the later part of the period when the information is more reliable. In Group II (children taken in charge on a mixed indication) the proportion amounts, in round figures, to 30 per cent, and is thus lower. The same applies to Group III (seriously delinquent children) in which about $^1/_4$ of the fathers were sentenced during the later period. The lowest figure, about 20 per cent, are obtained for Group IV (young criminals). A comparison of the boys and the girls shows that the figures are fairly uniformly lower for the girls. The difference is not, however, statistically significant.

7.4 per cent of the mothers were sentenced which is 5 times as many as in the general population according to Dahlberg. The girls are more heavily handicapped in this respect than the boys, more than every 10th girl (11.6 per cent) and more than every 20th boy (6.1 per cent) had a mother who had been sentenced.

In so far as the frequency of inebriety after 1926 is concerned it is found that 22 per cent of the boys' fathers and 28 per cent of the girls' fathers were the objects of measures by

the Temperance Board. The figures are highest for Groups I and II, where the fathers of the boys in nearly 50 per cent respectively nearly 30 per cent were the objects of such measures. For Groups III and IV the corresponding figure is about 20 per cent.

According to the available information sexual promiscuity could be shown to be practised by the mothers of over $^1/_3$ of the boys in Group I but in the other groups the figures are considerably lower. The mothers of the girls practised sexual promiscuity to a somewhat greater extent than those of the boys but the difference is not statistically significant.

It is further shown that a disproportionately high percentage of the children taken in charge belonged to families who had immigrated into Malmö. This is specially the case for the girls.

III. THE MAIN INTEREST IS ATTACHED TO THE INVESTIGATION OF THE SOCIAL PROGNOSIS OF THESE CHILDREN WHEN THEY BECOME ADULTS.

This has been established by means of calculations of the annual risk and the total risk up to various ages. This has been done in respect of criminality (crimes which led to the inclusion of the perpetrator on the Central Penal Register), inebriety (measures by the Temperance Board and offences of drunkenness) and whether the persons concerned were the objects of measures against vagrancy or received public assistance.

The annual risk and the total risk up to various ages were calculated from the time when the children were discharged by the Child-Welfare Board, or in the case of Group IV the time of their dismissal by the court or their release from detention homes or prisons. For those belonging to Group I, who were taken in charge on account of bad home conditions, the figures for risk have been calculated from the 16th birthday.

Some of these children (Group I), however, manifested delinquency before they reached this age, i.e. $^1/_3$ of the boys and not quite $^1/_5$ of the girls. From amongst these 5 per cent of the total number of boys and 9 per cent of the total number of girls in the group were transferred to a detention home.

8) It is of considerable interest to compare the different

groups in regard to their *social prognosis*. That is, to compare the prospects for those coming from bad homes without being delinquent (Group I), with those for the less and more seriously delinquent children (Groups II and III) and young criminals (Group IV). In this way a certain insight is gained into the significance of home conditions in comparison with that of other factors, even though the interpretation of the figures is by no means unequivocal. The figures, have, in any case, a practical value, and should provide a certain basis for theoretical discussion which are chiefly concerned with the relative importance of heredity and environment.

The main result may be seen in Figs. 11—17. A table is further included here giving the percentages of men who committed the misdemeanours mentioned above before the age of 40 years. For comparative purposes the corresponding figures for a cross-section of the population are also given.

Group	I	II	III	IV	Normal population
Criminality	24.3 ± 4.2	36.6 ± 5.8	47.1 ± 3.3	51.2 ± 4.1	7.7 [1]
Alcoholism	11.4 ± 3.8	20.5 ± 6.0	24.5 ± 3.1	23.4 ± 2.9	4.3 [2]
Vagrancy.....	7.9 ± 2.5	6.0 ± 2.8	7.0 ± 1.3	7.0 ± 1.3	0.6 [3]
Poor relief	37.2 ± 5.5	47.2 ± 7.0	51.9 ± 3.9	45.8 ± 3.5	

[1] Malmö 1937.
[2] Malmö 1929—33.
[3] The whole country.

It is thus shown that the prognosis is best in Group I. Then come Groups II and III. The prognosis in Group IV is also bad. In order to clarify the situation further it may be mentioned that, if those who were not convicted of crime, nor taken in charge by the Temperance Board nor warned against vagrancy are considered as blameless, about $^2/_3$ (70 per cent) of the members of Group I escaped being the objects of such measures before the age of 40 years. The corresponding figure for Group II is 49 per cent, while that for Groups III and IV is barely 43 per cent respectively 41 per cent.

The material is not so large concerning women. The corresponding figures for them up to the age of 35 years are as follows:

Group	I	II	III	IV	Normal population
Criminality	5.2 ± 1.9	11.7 ± 5.5	21.1 ± 4.3	37.3 ± 13.7	0.8[1]
Vagrancy.....	2.0 ± 1.5	13.6 ± 5.9	16.8 ± 3.5	9.6 ± 7.9	0.14[2]
Poor relief	11.0 ± 2.7	18.0 ± 6.1	33.3 ± 4.8	21.8 ± 8.9	

[1] Towns 1937.

[2] The whole country 1932.

The figures are considerably more favourable than for men but otherwise they show the same tendency, i.e. they are lowest for Group I, considerably higher for Group II and highest for Groups III and IV. The difference from the normal population is specially conspicuous. In regard to the frequency of blameless individuals among the women at 35 years of age (if the same principle is followed as above for the men which in this case seems appropriate), we find, on the whole, the same trend of the figures as for the men. The figures for the women, however, are much higher than those for the men of corresponding ages. The difference between Groups I and II is not so marked for the women as for the men. The frequency of blameless women at 35 years of age in the different groups is: Group I, 94 per cent, Group II, 83 per cent, Group III, 74 per cent and Group IV, 58 per cent.

9) Finally the material has been used to investigate the effect of certain special factors. The number of cases in the different groups was too small to allow of a comparison being made. A weighted percentage based on a proportionate representation of the groups compared has therefore been used.

a) If legitimate and *illegitimate* children are compared in this way no significant difference in prognosis is found in the matters towards which the investigation was directed.

b) If the children of *criminal parents* are compared with those whose parents are not criminals it is found that the child-

ren of criminal parents themselves become criminals somewhat more frequently than other children. (The difference is statistically probable.)

c) If the material is divided according to whether the *fathers were drunkards* or not it is found that the children of drunkards are more liable to become criminal than other children. The investigation, however, provides no proof that the children of drunkards themselves become drunkards more frequently than other children.

d) The *home conditions* appear to have a certain effect on the social prognosis of the children. Thus children from bad homes more frequently become criminals (statistically probable difference) or drunkards or require public assistance when compared with children from good homes.

e) It may finally be stated that the investigation did not show that the *degree of intelligence*, the length of the period of *detention* or the *age* at which they were taken in charge or at which they first came into *contact* with the social authorities had any significant influence on the children's later social development. As the number of cases is not large in regard to the problem mentioned under 9), it is quite possible that differences might be found on a larger material. If the differences, however, had been very large, they would have appeared.

LITERATURE CITED.

AHNSJÖ, SVEN. 1941. Delinquency in girls and its prognosis. Lund.

ARCTANDER, SIGNY & DAHLSTRÖM, S. 1932. Hvordan går det våre vergerådsbarn? (En statistisk studie bygget på erfaringer fra Oslo vergeråd 1900—1928.) Oslo.

ARCTANDER, SIGNY. 1936. Vergerådsopdragelsen og dens resultater. Statsøkonomisk Tidsskrift. Hefte 3—5. Oslo.

BAER: Quoted from GRUHLE.

BARTH, E. 1915. Untersuchungen an weiblichen Fürsorgezöglingen. Ztschr. f. d. gesamte Neurologi und Psychiatri. Berlin Bd. 30.

BRANDER, T. 1936. Studien über die Entwicklung der Intelligenz bei frühgeborenen Kindern. Diss. Helsingfors.

— 1938. Über zerebral defekte Kaiserschnittkinder. Acta pæd. Bd. 23 H. 2.

BRONNER, AUGUSTA. 1914. A Research on the Proportion of Mental Defectives among Delinquents. Journ. of. Crim. Law and Crim. Vol. 5. American Instit. of criminal law and criminalogi.

— 1938. Delinquency and Feeblemindedness. Festskrift tillägnad Olof Kinberg. Stockholm.

BURT, CYRIL. 1925. The young Delinquent. London.

Centralförbundet för socialt arbete. 1941. Ligabrottsligheten. Stockholm.

— 1942. Ungdomsbrottsligheten. Social årsbok. Stockholm.

Child Guidance Center: Quoted from BRONNER (1938).

CREMIEUX, ALBERT, SCHACHTER ET COTTE, S. 1945. L'enfant devenu délinquent. Marseille.

DAAE, A. 1908. De samlede Resultater av de norske Gutte-Skolehjems Virksamhet i Tidsrummet 1/1 1901—31/12 1907. Nordisk Tidsskrift for Fængselvæsen. Aarg. XXXI. Nr 31.

DAHLBERG, GUNNAR och STENBERG, SVEN. 1934. Alkoholism ss samhällsproblem.

DAHLBERG, GUNNAR. 1937. Sjukdomarna och samhället.

— 1937. On the Frequency of Mental Deficiency. Upsala Läkareförenings förhandlingar. Ny följd. Bd. XIII. H. 5—6. Uppsala.

— 1939 b. Alcoholism from a Social Point of View. A survey of conditions in Stockholm. Acta Soc. Med. Suec. Bd. 65.
— 1940. Statistical Methods for Medical and Biological Students. London.
— 1942. The occurrence of alcoholics and their treatment. A social-statistic analysis for the town of Malmö 1929—1938.
— 1943. Risken att dömas för svårare brott. Nordisk Tidskrift for Strafferet. Hf. 3.
— 1944. Utredningar rörande fylleriförseelser och alkoholism. Särtryck ur Statens off. utr. 1944: 3.
— 1946. Om risken för äktenskap och skilsmässa. Social-Medicinsk Tidskrift. Hf. 5.

DAHLBERG, GUNNAR och LINDBERG, TORGNY. 1944. Om frekvensen av återfall i brott. Särtryck ur Statens offentl. utredn. 1944: 50.

Fattigvårdslagstiftningskommitténs betänkande III. 1915. Lagstiftning rörande minderåriga förbrytare. Förslag till lag om ändring av 5 kap. 3 § strafflagen. Stockholm.

Federal Childrens Bureau: Quoted from BRONNER (1938).

FREY, TORSTEN S:SON. 1944. Om vanart hos flickor i Stockholm. Sv. Läkaretidn. Hft. 2.

FRITZELL, Y. 1935. Redogörelse för en statistisk undersökning rörande avgångna skyddshemselever med avseende på deras uppförande efter utskrivningen. Meddelande fr. Kungl. Socialdep. byrå för fattigvårds- och barnavårdsärenden. N:r 59. Stockholm.

FUCHS-KAMP, ADELHEID. 1929. Lebensschicksal und Persönlichkeit ehemaliger Fürsorgezöglinge. Berlin.

Fångvårdsstyrelsen. 1942. Utredning ang. ungdomsbrottsligheten.

GLUECK, S. & GLUECK, ELEANOR. 1930. 500 Criminal Careers. New York.
— 1934. One thousand juvenile delinquents. Cambridge Mass.
— 1945. After-Conduct of Discharged Offenders. London.

v. GRABE, E. 1923. Spätschicksale von Fürsorgezöglingen und Prostituierten. Arch f. Kriminol. Bd. 75.

GREGOR, A. 1918. Die Verwahrlosung ihre klinisch-psychologische Bewertung und ihre Bekämpfung. I Teil: Die Verwahrlosung der Knaben.
— 1932. Ergebnisse und zeitgemässige Aufgaben der Antaltfürsorgeerziehung. Ztschr. f. Kinderforschung 40 Bd. Hf. 3. S. 291.

GREGOR, A. & VOIGTLÄNDER, ELSE. 1918. Die Verwahrlosung, ihre klinisch-psychologische Bewertung und ihre Bekämpfung. II Teil: Die Verwahrlosung der Mädchen.

GRUHLE, H. W. 1912. Die Ursachen der jugendlichen Verwahrlosung und Kriminalität. Berlin.
— 1938. Seelische Folgen von Kopfverletzungen. Med. Klin. II. S. 928.

HEALY, W. 1924. The Individual Delinquent. Boston.

HEALY, W. & BRONNER, AUGUSTA. 1926. Delinquents and Criminals. Their Making and Unmaking. Studies in Two American Cities. New York.

HEALY, W., BRONNER, AUGUSTA F., BAYLOR, EDITH M. H. & MURPHY, J. P. 1931. Reconstructing Behaviour in Youth. A Study of Problem Children in Foster Families. New York.

HERY: Quoted from GRUHLE.

HOLMSTEDT, GUSTAF. 1944. De utomäktenskapliga barnen. Tidskrift för barnavård och ungdomsskydd N:r 2.

HYRENIUS, HANNES. 1943. Studier rörande den utomäktenskapliga fruktsamhetens variationer. Skrifter utgivna av Fahlbeckska stiftelsen nr. 28.

INGHE-JONSSON. 1942. Social conditions ond complex of motives of the abortion patients. Acta Obstetr. et Gyn. scand. Vol. XXII.

INGHE, GUNNAR. Mental abnormities among criminals. Acta Psych. et Neurol. Vol. XXVI.

JACOBSEN, S. 1922. Barnekriminaliteten i Kristiania. Barn och ungdom. Nordisk Social-Pædagogisk Tidsskrift. Oslo.

JONSSON, GUSTAV. 1944. Undersökningar angående vanartiga barn och asocial ungdom. Bilaga till Ungdomsvårdskommitténs betänkande ang. Psykisk Barn- och Ungdomsvård.

KEMP, T. 1936. Prostitution: An investigation of its causes, especially with regard to hereditary factors. Copenhagen.

KLUGE, O. 1926. Zur Erfolgsstatistik abnormer Fürsorgezöglinge. Psychiatr. Neur. Wochenschr. Bd. 28.

KNÖS, HELGE. 1938. Till frågan om ungdomskriminalitet i Sverige. Socialmed. tidskrift ht. 6.

VON KOCH, RAGNAR. 1939. Samhällets barnavård. Stockholm.

LANGE, JOHANNES. 1928. Verbrechen als Schicksal. Studien an kriminellen Zwillingen. Leipzig.

LIEPMANN: Quoted from GRUHLE.

LINDHEIM: Quoted from GRUHLE.

LUND, DAVID. 1918. Die Jugendasozialität. Uppsala.

LÜCKERATH. 1934. Erfolge in der Fürsorgeerziehung. Zeitschr. f. Kinderforschung. Bd. 43 Hf. 1. s. 36.

MÖNKEMÖLLER, O. 1911. Zur Kriminalität des Kindes Alters. Archiv f. Kriminalanthrop. und Kriminalistik. Bd. 40.

— 1916. Die kindliche Kriminalität in der Fürsorgeerziehung. Vierteljahrschr. gerichtl. Med. Bd. 52.

NISSEN, H. 1917. Forsømte og forbryderske barn i Norge. (Enfants moralement abondonnés et enfants criminels en Norvège.) Norges officielle Statistik VI 99.

NORDAL, G. 1935. En medicinsk-psykologisk undersökning av elever vid vissa skyddshem. Meddelande från Kungl. Socialdepartementets byrå för fattigvårds- och barnavårdsärenden. Nr 59. Stockholm.

OHLAND, ANNALIESE. 1937. Der Erfolg der deutschen Fürsorgeerziehung. Deutsche Jugendhülfe. Berlin n:r 1 o. 2.

OWENS, ALBERT A. 1929. The behaviour Problem Boy. A social-educational Survey. Philadelphia.

POLLAK: Quoted from GRUHLE.

REGNÉR, ELIS. 1943. Vanart bland barn och ungdom. Social-med. Tidskr. hf. 2.

REHM, NN. 1926. Das soziale Schicksal psychopatischer Fürsorgezöglinge. 2 Neur. 106. Hf. 4, 5.

ROBISON, SOPHIE MOSES. 1936. Can Delinquency be measured? Columbia University Press. New York.

ROSANOFF, A. J. 1932. A study of mental disorders in twins. Evgenical News. Vol. XVII.

ROSANOFF, A. J., HANDY, L. M. & ROSANOFF, I. A. 1936. Criminality and Delinquency in Twins. Journ. of Crim. Law and Crime. vol. 24.

SCHOTT: Quoted from GRUHLE.

SCHWALBE-HANSEN, P. A. 1938. Efterundersøgelse af »psykopatiske» Børne. Hospitalstidende. Bd. 181. nr. 17.

SHAW, CLIFFORD R.: Quoted from ROBISON.

SKJERBÆK, O. J. 1929. Om vore værgeraadsbørn og deres videre Skæbne. Forhandl. paa Dansk Kriminalistforenings to og tyvende Aarsmøde. København.

Sociala meddelanden. 1924. Undersökning rörande manliga tvångsarbetare i Sverige år 1923.

SPANN: Quoted from GRUHLE.

SPÄTH, ALFRED. 1934. Ergebnisse der Fürsorge-Anstaltserziehung. Zeitschr. f. Kinderforschung 43 Bd. Hf. 1. S. 42.

STARKE: Quoted from GRUHLE.

Statens offentliga utredningar. 1944. Justitiedepartementet. Ungdomskommitténs Betänkande med utredning och förslag ang. Psykisk Barna- och Ungdomsvård.

SUNDBERG, EMIL. 1903. Lagstiftningen om Barnavård. Stockholm.

Sveriges Officiella Statistik. 1928, 1931, 1934, 1937, 1943. Samhällets barnavård.

Sveriges Officiella Statistik. 1933—36, 1929—32, 1921—32. Riksdagsmannavalen.

THOMA: Quoted from GRUHLE.

TRASHER, F. H. 1937. The Gang. Chicago.

TÖBBEN, H. 1927. Die Jugendverwahrlosung und ihre Bekämpfung. Münster.

Ungdomsvårdskommitténs betänkande I. 1944. Med utredning och förslag ang. Psykisk Barna- och Ungdomsvård. Stockholm.

WAGNER, G. 1936. Kriminalität und Prostitution ehemahliger weiblicher Fürsorgezöglinge — ein Beitrag zum Problem »Erfolg der Fürsorgeerziehung». Monatschr. f. Krim. psych. u. Strafrechtsreform. Jahrg. 27. H. 10.

WHITE, INGER. 1940. Redogörelse för en undersökning rörande skyddsuppfostrade flickor, som åren 1931—1939 intagits på Fredrique Wohlfarts Stiftelse. Tidskrift för Barnavård och Ungdomsskydd. Årgång 15. nr. 3.

WIMMER, AUG. 1929. Om vore Værgeraadsbørn og deres videre Skæbne. Forhandl. paa Dansk Kriminalistforenings to og tyvende Aarsmøde. København.

ZIEHEN, TH. 1926. Die Geisteskrankheiten des Kindesalters. Einschliesslich Schwachsinn und Psychopatische Konstitutionen. 2 Aufl. Berlin.

CONTENTS.

	Page
Preface	IX
Chapter I. Survey of literature	1
Frequency	2
Age	6
Types of delinquency	9
Causes of delinquency	18
Illegitimacy	19
Broken homes	26
Economic factors	33
Alcoholism	35
Criminality of the parents	39
Immorality in the parents	41
Environmental conditions outside the home	44
Qualities in the parents	48
Qualities in the children	53
Prognosis	66
Chapter II. Description of the material	93
Chapter III. The incidence of delinquency	99
Chapter IV. Age when taken care of	104
Chapter V. Types of delinquency	117
Chapter VI. Home conditions during childhood	135
Illegitimate birth	140
Broken homes	145
Cohabitant parents	150
Change of home	151
Social position of the family	151
General character of the home	156
Financial factors	164
Siblings	168
Chapter VII. Characters of the parents	173
Abuse of alcohol among the parents	179
Chapter VIII. Distribution of the homes on different regions of the town	185
Chapter IX. Frequency of immigrants in Malmö in this material	188
Chapter X. State of health of the children	194
Chapter XI. Mental character of the children	199
Intellectual equipment	199
Frequency of temperamental abnormalities among children	211

Chapter XII. Measures taken by the authorities 215
Length of the period under care 219
Chapter XIII. Prognosis 225
Introduction 225
Conduct during the time after being taken in charge and before the age of 15 years for children coming from bad homes (Group I) 233
Prognosis of children from bad homes (Group I) 236
1. Prognosis as regards criminality 237
2. Prognosis as regards temperance 245
3. Prognosis as regards vagrancy 247
4. Prognosis as regards self-support 248
5. Social position 250
Prognosis of slightly delinquent children from bad homes (Group II) 253
1. Prognosis as regards criminality 254
2. Prognosis as regards temperance 258
3. Prognosis as regards vagrancy 258
4. Prognosis as regards self-support 259
5. Social position 260
Prognosis regarding more serious cases of delinquency (Group III) 260
1. Prognosis as regards criminality 260
2. Prognosis as regards temperance 266
3. Prognosis as regards vagrancy 267
4. Prognosis as regards self-support 267
5. Social position 268
Prognosis as regards criminal juveniles (Group IV) 269
1. Prognosis as regards criminality 269
2. Prognosis as regards temperance 274
3. Prognosis as regards vagrancy 275
4. Prognosis as regards self-support 276
5. Social position 276
Chapter XIV. Comparison between the different groups from prognostic aspects 277
Chapter XV. Prognosis in regard to certain special characters 288
Introduction 288
Prognosis and illegitimacy 289
1. Illegitimacy and criminality 289
2. Illegitimacy and inebriety 291
3 and 4. Illegitimacy and vagrancy and poor relief 292
Prognosis and home conditions 293
1. Home conditions and criminality 294
2. Home conditions and inebriety 295
3. Home conditions and vagrancy 296
4. Home conditions and poor relief 296
Prognosis and intellectual level 297
1. Intellectual level and criminality 297

2. Intellectual level and inebriety 299
3. Intellectual level and vagrancy 299
4. Intellectual level and poor relief 299
Qualities of the parents and prognosis of the children 301
1. Criminality of the parents and the children 301
2. Abuse of alcohol by the fathers and the prognosis of the children 302
Prognosis as regards criminality 303
Prognosis as regards inebriety 304
Criminality and length of detention 305
Criminality in relation to the age at which the child was taken in charge 306
Summary 311
Literature cited 319

DISTRIBUTION OF THE HOMES ON DIFFERENT REGIONES OF THE TOWN
o Dwellings of children from bad homes.(Group I)
• " " delinquent children and criminal youths (Groups II,III,IV)
N
S
0
500
1000 m

UNIV. OF MICH.

FROM THE PEDIATRIC CLINIC OF KAROLINSKA INSTITUTET AT KRONPRINSESSAN
LOVISA'S CHILDRENS HOSPITAL, STOCKHOLM.
HEAD: PROFESSOR A. LICHTENSTEIN.

GALLOP RHYTHM IN CHILDREN

STUDIED BY MEANS OF CALIBRATED PHONOCARDIOGRAPHY

AN EXPERIMENTAL AND CLINICAL INVESTIGATION

BY

LARS-ERIK CARLGREN

Acta Pædiatrica. Vol. XXXIII. Supplementum VI.

LUND
HÅKAN OHLSSONS BOKTRYCKERI
1946

Contents

Preface 5

Chapter I. Survey of the literature 7

The third heart sound 7

The auricular sound 13

The gallop rhythm 18

Summary 35

Chapter II. Methods 38

Apparatus 38

Statistical methods 41

Chapter III. Experimentally produced gallop rhythm 43

Earlier investigations regarding myocarditis after coffein-adrenalin injections 43

Material and methods 46

The normal phonocardiogram of rabbits 47

Changes in the hearts of rabbits after coffein-adrenalin injection 48

Gallop rhythm at experimentally produced myocardial damage in rabbits 57

Summary 61

Chapter IV. The normal phonocardiogram in children 63

Description of normal cases 63

Analysis of the normal phonocardiogram in children 64

Summary 77

Chapter V. Definition of gallop rhythm in children by means of calibrated phonocardiography 79

Chapter VI. Gallop rhythm in childhood. Analysis of 104 cases .. 81
Introduction 81
Gallop rhythm in rheumatic infection 86
Gallop rhythm in other infections 98
Gallop rhythm in congenital heart disease 100
Gallop rhythm in anemia 101
Gallop rhythm, other cases 101
Discussion 110

Results of the investigation 112

Literature 118

Appendix 125

Case history 127

When nothing else is mentioned the phonocardiograms and electrocardiograms presented in the figures are reproduced in half scale.

PREFACE

Auscultation has, ever since the days of LAËNNEC, been considered one of the most useful diagnostic means in cardiology. The principal attention has of old been directed to the two ordinary heart sounds and the murmurs, the hearing capacity of the ear often being to some extent overestimated. Thus, faint systolic murmurs, although as shown by later investigations a normal occurrence, have frequently been erroneously interpreted as pathological. On the other hand, particularly in the case of children, very little notice has been paid to the gallop rhythm. Here phonocardiography opens up new prospects of objective registration which is all the more important since gallop sounds are to be found principally in the lower frequency ranges of the tonal scale where the sensitivity of the ear is low.

The gallop rhythm is due to an accentuation of normal sound phenomena which only exceptionally are distinctly audible, but to a great extent are registered in quite healthy individuals. The difference between gallop sounds and the corresponding physiological extra sounds has earlier been vaguely defined. This difference can be expressed in figures by means of calibrated phonocardiography, in accordance with MANNHEIMER's (1940) observation that the gallop sounds have a higher frequency and larger amplitude than the normal third sound and auricular sound, respectively.

The purpose of the present investigation was to produce an objective definition of the gallop rhythm on the basis of which a study of the occurrance and significance of this symptom in childhood could be performed. In addition, an experimental examination has been carried out in order to elucidate more closely the mechanism of origin.

The greater part of the investigation has taken place at the Pediatric Clinic of the Crown Princess Lovisa's Children's

Hospital in Stockholm. The author wishes to present his warm thanks to the head of this Clinic, Professor ADOLF LICHTENSTEIN, for the interest he has shown in this investigation, for his invaluable advice, and also for the special opportunities offered me to pursue this inquiry at his Clinic.

I am greatly indebted to Dr EDGAR MANNHEIMER — who is in charge of the Heart Station at the Clinic — for his suggesting this subject of research and for his generous advice and help from the beginning to the end of the investigation.

To Professor GUNNAR DAHLBERG I wish to express my sincere gratitude for his help with the statistical analysis and for the kind interest he has shown throughout.

The part of the investigation comprising the determinations of the heart volume was undertaken at Norrtull's Hospital, Stockholm. My thanks are due to Professor ARVID WALLGREN for the facilities rendered in this connection.

I am much obliged to Dr URBAN HJÄRNE, Chief Doctor of the Elementary Schools of Stockholm, for kindly placing at my disposal the children's health reports.

Dr OLIVER AXÉN has carried out the measurements which form the basis of the determinations of the heart volume. Dr PAUL KALLÓS has contributed his helpful advice in connection with the experimental examinations. The röntgenograms of the rabbit hearts have been studied together with Dr FOLKE ULFSPARRE, and Dr STIG RANSTRÖM has helped me in analysing the microscopical specimens. I wish to record my special thanks to all those mentioned here.

I owe a debt of gratitude to Miss ULLA MOBERG for the care and tireless attention she has devoted to this work. I am also indebted to Miss ULLA AUGUSTSSON for excellent secretarial assistance, to the Röntgen nurses EDITH NILSSON and VERA VIGANT and to Mrs ULLA SCHÖTT for her translation of this work.

The costs of the investigation have been partially defrayed by grants from the Therèse and Johan Andersson Fund.

Stockholm, in November 1946.

Lars-Erik Carlgren

CHAPTER I

Survey of the literature

The third heart sound

OBRASTZOW (1905) seems to be the first to have observed the physiological third heart sound. In his opinion, the proto-diastolic gallop rhythm is due to an accentuation of a normal accessory sound in diastole. However, GIBSON and HIRSCHFELDER are usually given the credit of being the first to have heard the physiological third sound independently of one another in the year 1907. About the same time, EINTHOVEN succeeded in registering vibrations at the beginning of diastole in a few of his phonocardiograms, the time of their appearance corresponding to that reported by GIBSON with regard to the third sound. The first, comprehensive, clinical description of this sound phenomenon was presented by THAYER in 1909. OHM (1921) demonstrated, by means of simultaneous phonocardiography and registration of the venous pulse, that the third heart sound appears at the end of the phase of rapid ventricular filling.

As a rule, the third heart sound is characterized (by THAYER 1909, and others) as a weak, barely perceptible sound of low frequency occurring at the beginning of diastole soon after the second sound. It is only, or best, audible in a supine position. However, it was also perceived by STEINBERG (1925) fairly often in an erect position. It is more distinct in a left lateral position than in a dorsal one, and the intensity increases at pressure over the epigastrium or at elevation of the legs. A certain degree of inconstancy in the appearance of this heart sound has been pointed out by several authors, such as THAYER (1909), GALLAVARDIN (1912), MELIK-GÜLNASARIAN (1932), and ROUTIER and VAN BOGAERT (1934 b). According to them, it is often perceptible but for a short while immediately after the examined person

has assumed a recumbent position, only to become fainter or disappear later. Even in cases where the sound remains, the intensity is generally not constant but varies with the respiratory phase, i. e. it attains its maximum during and towards the end of the expirium. The point of maximal audibility is, in the opinion of the majority of investigators, the apex or just medially and above it. ROUTIER and VAN BOGAERT (1934 b) state that a faint systolic murmur is frequently observed simultaneously with the third sound, appearing as inconstantly as this sound.

The third heart sound is registered phonocardiographically by one or, at most, a few vibrations at the beginning of diastole, soon after the second sound. Even in different individuals the interval between the two sounds varies within fairly narrow limits. It is practically constant in one and the same person and independent of the pulse frequency. The distance to the subsequent first sound, on the other hand, varies according to the duration of the cardiac cycle. As a rule, the interval from the beginning of the second sound is defined in the literature as amounting to 0.11—0.18 seconds.

There is a considerable divergency of opinion regarding duration and frequency. This is explained by ORIAS and BRAUN-MENENDEZ (1939) as due to the variety of methods employed. Thus, the duration is stated to equal between 0.02 and 0.14 seconds and the frequency between 25 and 50 cycles per second. For the same reason, the amplitudes given by various authors, with the exception of MANNHEIMER (1940), are not reproducible.

The amplitude in one and the same individual is generally not constant, but varies within different cardiac cycles, usually in relation to the respiration, i. e. increasing during the expirium and decreasing during the inspirium (ORIAS and BRAUN-MENENDEZ). The amplitude is also dependent on the point of application of the microphone. LEONHARDT (1932) found the largest amplitude in the 4th intercostal space in the left parasternal line. It decreased considerably even at but a slight deviation of the microphone sidewards. CAEIRIO and ORIAS (1937) observed no definite variation in the amplitude at registration over apex. the mesocardium and the pulmonary artery. However, the

material at their disposal was fairly restricted. MANNHEIMER (1940), on the other hand, was able to ascertain a larger amplitude of the third sound at the apex than at the pulmonary artery, the difference being statistically significant. The position of the body also plays a part in this connection. MANNHIEMER observed lower amplitudes in an erect than in a supine position, the difference being, as above, statistically significant.

The etiology of the third heart sound.

Several theories have been propounded regarding the cause of the physiological third heart sound. According to EINTHOVEN (1907), it is due to vibrations transmitted from the aortic valves to the apex. GIBSON (1907) and HIRSCHFELDER (1907) state that the third sound originates in the rapid and transient closure of the auriculo-ventricular valves after the quick ventricular filling in diastole. This opinion was shared by GALLAVARDIN (1912) and BRIDGMAN (1915). An opposite view is held by THAYER (1909) who maintains that the third sound arises from the sudden opening of the same valves at the beginning of diastole. However, DEAN (1916) was able to show, in experiments on cat hearts, that no movement of the auriculo-ventricular valves takes place during the phase of diastole when the third sound occurs. By means of simultaneous registration of the heart sounds and the venous pulse, OHM (1921) and later BRAUN-MENENDEZ and ORIAS (1934) proved that the third heart sound coincides regularly with the descending line of the *v* wave and, accordingly, arises at the end of the phase of rapid ventricular filling. OHM explains the origin of the third sound, i. e. »der diastolische Ruck», as due to the sudden stemming of the precipitate inrush of blood by the ventricular wall at the beginning of diastole. It is, moreover, nowadays generally interpreted as a muscular sound (e. g., GUBERGRITZ 1925, 1929, MELIK-GÜLNASARIAN 1932, SCHÜTZ 1933, BRAUN-MENENDEZ and ORIAS 1934), this conception deriving support from a number of experimental works. LEONHARDT (1932), for instance, performed experiments on dogs, exposing the ventricular wall and making it serve as one of the condensator plates in a high frequency oscillating

circuit. He was thus able to find vibrations of the ventricular wall coinciding with the third heart sound. BOYER (1942) showed that a very faint tap by mechanical means against the ventricular wall of a heart which has stopped beating causes a sound much resembling the normal third sound as regards frequency, amplitude and duration. SMITH (1944) carried out the following experiment on heart and lung preparations from dogs. He stopped the blood flow to the heart, simultaneously immobilizing the valves between the ventricle and the auricle. This caused a disappearance of the second sound as soon as the heart became empty of blood. The first and third sounds, on the other hand, showed practically no changes at registration, so long as the heart made the slightest contraction. This way SMITH considers himself to have proved that at least the main part of the third sound originates from vibrations in the ventricular wall.

The vibrations causing the third heart sound are, in the opinion of most investigators, due to the rapid inrush of blood from auricle to ventricle at the beginning of diastole. The intensity of the third sound is therefore, according to ORIAS and BRAUN-MENENDEZ (1939), influenced by the degree of suddenness with which the blood rushes into the ventricle, on the one hand, and by the muscular tone and the reaction against the inrushing stream of blood connected with it, on the other. The first-mentioned factor, viz., the degree of suddenness of the inrushing blood, is related to the pressure difference between the auricle and the ventricle at the particular moment when the auriculoventricular valves are opened. This pressure difference is, under normal circumstances, greatest on the very occasions when the third sound is the most distinct, i. e. at the change from an erect to a supine position, at elevation of the legs or at pressure on the epigastrium. The effect of the muscular tone on the intensity of the third sound should, above all, apply to pathological conditions. In the case of myocardial damage, the tone of the ventricular wall is often considerably reduced, which would offer prerequisites for a pathological accentuation of the third sound.

The significance of the vegetative nervous system and, above all, that of the vagus with regard to the occurrence of the

physiological third sound has been emphasized by several authors. As early as in 1905, OBRASTZOW perceived it chiefly in neurasthenic individuals. GUBERGRITZ (1925) often came across a third sound during convalescence after typhus fever and relapsing fever, which had not been present during the acute stage of the disease with the more rapid heart rhythm. He attributed it to an increased vagal tone. WIKNER (1930), who had practically never heard a third sound in fully normal hearts, noticed it in several cases of cardiac neurosis. MELIK-GÜLNASARIAN (1932) perceived this sound principally in anemic, neurasthenic and convalescing individuals. According to ROUTIER and VON BOGAERT (1934 b), the high frequency of the third sound in children might be explained by the hypertonia of the vagus which is physiological at this age. MELIK-GÜLNASARIAN also performed experiments on dogs in order to study the relation between the tone of the cardiac muscle and the third heart sound. At a moderate stimulation of the vagus by means of electric current or drugs, slower and stronger contractions of the heart were obtained as well as an increased diastolic dilatation, simultaneously with an accentuation of the third sound and its consequent audibility in a larger number of cases. On the other hand, an increased vagus stimulation involved weaker contractions and a further dilatation, causing the third sound to become fainter and finally to disappear. At tachycardia, owing to paresis of the vagus or stimulation of the sympathetic nerve, no third sound was perceived in the majority of cases. The heart disclosed marked contractions but insufficient diastole which caused a strong decrease in volume. Thus, a simultaneous increase of the systolic tone and decrease of the diastolic tone form, according to MELIK-GÜLNASARIAN, the best prerequisites for an appearance of the third heart sound.

The occurrence of the third heart sound.

The third sound has been perceived to a greatly varying extent by different authors. It is evident that, apart from training and experience, also the hearing capacity of the investigator has been of great significance in this respect.

TABLE 1.

Frequency of third sound at auscultation (Thayer 1909).

Age in years	1—10	10—20	20—30	30—40	40—50	50—60
Number of examined persons	39	90	55	26	14	7
Percentage frequency of third sound	58.9	84.4	50.9	42.3	14.0	0

TABLE 2.

Frequency of third sound at phonocardiographic registration (Frost 1944).

Age in years	10—19	20—29	30—39	40—49	50—80
Number of examined persons	26	40	34	30	39
Percentage frequency of third sound	35	18	3	3	0

TABLE 3.

Frequency of third sound in children according to different authors.

Author	Age distribution	Number of cases	Percentage frequency of third sound at	
			Auscultation	Phonocardiography
Bridgman (1915)	12—15 years	16	81	100
Steinberg (1925)	4—14 »	100	48	
Leonhardt (1932)	3—14 »	41		84
Mc Kee (1938 a)	5—17 »	105	6	66
Mannheimer (1940)	0—14 »	135		69.6±4.0

THAYER (1909) heard a third heart sound in 65 per cent of healthy persons under the age of 40 years. The frequency at various ages is illustrated in Table 1 below.

Contrary to THAYER, GALLAVARDIN (1912) was able to hear a physiological third sound only in rare instances. OBRASTZOW (1919) and GUBERGRITZ (1925) found it in approximately 90 per cent of persons with normal hearts at direct auscultation with-

out a stethoscope. The latter was, moreover, of the opinion that the complete absence of a third sound indicates a pathological change in the myocardium.

As already mentioned, EINTHOVEN (1907) was the first to ascertain the normal third sound by an objective method. Since then, a number of phonocardiographic investigations have been published, the third heart sound being registrable in a great percentage of cases. Thus, BRAUN-MENENDEZ and ORIAS (1934) were able to detect it in 42 out of 100 healthy medical students, and CAEIRIO and ORIAS (1937) in 65 per cent of a similar material by means of a more sensitive method. PEREIRA (1939) registered it in 51 per cent of 49 healthy women. Only FROST (1944) seems to have compared the frequency of the third sound at different ages (see Table 2).

Thus, it is apparent that a registrable third sound is a normal and frequent phenomenon in young individuals. On the other hand, as demonstrated by FROST, it gradually becomes more rare with increasing age. As is shown in Table 3, it has been registrable in children to a very large extent. The audibility has been judged very differently by various authors also in this connection.

The auricular sound

As early as in 1840, CLENDINNING established the fact that the auricular contraction could give rise to a faint sound. Furthermore, in dealing with the question of gallop rhythm, POTAIN (1875) described a physiological sound appearing at the point of the cardiac cycle where the presystolic gallop rhythm is usually heard. The physiological auricular sound has otherwise almost without exception escaped notice at auscultation. This is easily understood since it is in itself very faint and of low frequency, on the verge of audibility and, in addition, appears in immediate proximity to the strong first heart sound. The first comprehensive phonocardiographic investigations of the auricular sound were performed by BRIDGMAN (1914) and BENJAMINS (1914). The latter registered the heart sounds via the oesophagus and was able to ascertain a distinct auricular sound in

all the examined cases. His results have been confirmed later by TAQUINI and BRAUN-MENENDEZ (1935).

The auricular sound is registered phonocardiographically by one or two slow vibrations after the P wave in the electrocardiogram at a distance from the summit of the P wave of, on an average, 0.043 seconds (ORIAS and BRAUN-MENENDEZ 1939), and, from the beginning of the P wave, of 0.123 ± 0.003 seconds (MANNHEIMER 1940). The duration is, according to CAERIO and ORIAS (1937), 0.08—0.10 seconds and the frequency about 29 cycles per second.

BRIDGMAN (1914) states that changes of position, pressure over the epigastrium, etc., have the same effect on the amplitude of the auricular sound as on the third sound. MANNHEIMER (1940) also finds, as in the case of the third sound, a larger amplitude in a supine than in an erect position. CAERIO and ORIAS (1937) point out that the auricular sound is best registered at application of the microphone to the mesocardium. However, the differences given by them are not statistically significant. MANNHEIMER found a larger amplitude at the apex than over the pulmonary artery, with a statistically significant difference.

The etiology of the auricular sound.

KREHL (1889) was able to hear an auricular sound from the dying hearts of animals in which the ventricles had stopped contracting while the auricles continued to beat. HÜRTHLE (1895), who was the first to adopt a phonocardiographic method, registered vibrations before the first sound which he attributed to the auricular contraction. According to KAHN (1910), these vibrations occurred too long after the P wave in the electrocardiogram to be derivable from the muscular contraction itself. He assumed that they must instead be connected with the inrush of blood from the auricle to the ventricle. BRIDGMAN (1914) supposed the auricular sound to be due to vibrations in the ventricular wall when the blood was suddenly pressed down into the ventricle owing to the auricular contraction. Later, more definite information has been obtained regarding the mechanism of origin of this sound through a study of certain pathological

conditions and, above all, that of complete auriculo-ventricular block, as well as through the registration of the heart sounds via the oesophagus.

Although the last-mentioned method of investigation had been employed far earlier, BENJAMINS (1914) was the first to ascertain, in a number of healthy persons, the normal auricular sound in all the examined cases. TAQUINI and BRAUN-MENENDEZ (1935) and TAQUINI (1937) registered the heart sounds via the oesophagus as well as the precordium. The former subjected 15 healthy persons to examination by means of the phonocardiogram, the electrocardiogram and the phlebogram. A distinct presystolic sound was registrable in all the cases. At application via the oesophagus, it appeared 0.04—0.05 seconds after the beginning of the P wave and coincided, in the majority of cases, with the *a* wave in the phlebogram. It was invariably clearly perceived at auscultation and the rhythm mostly resembled a slow gallop. At registration over the precordium, the auricular sound did not appear until 0.04 seconds or more after the beginning of the *a* wave. When audible, it was mostly recognized as the initial portion of a duplicated first sound. The result of the investigation would, according to the authors, indicate that the sound which was registered via the oesophagus originated from the actual auricular contraction, while that which was registered over the precordium was presumably due to vibrations in the blood or in the ventricular wall owing to the rapid flow of blood from auricle to ventricle during auricular systole. TAQUINI (1937) confirmed these results on the basis of a larger material.

It has long been known that a faint sound may sometimes be perceived at regular intervals during ventricular diastole in cases of auriculo-ventricular block with complete dissociation between auricle and ventricle. Simultaneous registration by means of the phonocardiogram, the electrocardiogram and the venous pulse has shown that the sound is caused by auricular contraction. LEWIS (1915) was at times able to register a duplicated auricular sound in complete auriculo-ventricular block. He assumed that the first component was due to the muscular contraction of the auricle while the second component originated from the closure of the auriculo-ventricular valves after the

termination of the blood flow from auricle to ventricle. SELENIN and FOGELSON (1927) and FOGELSON (1933) shared LEWIS' opinion of the auricular sound as consisting of a muscular and a valvular part. BRAUN-MENENDEZ and SOLARI (1936) studied the auricular sound at complete block, produced experimentally on dogs. When exploring the auricle, it was registered 0.04—0.06 seconds after the beginning of the P wave. Two groups of vibrations were registered over the ventricles, one 0.06—0.15 seconds and the other 0.18—0.23 seconds after the beginning of the same wave. Thus, according to these authors, the auricular sound consists of three components, one being registered only over the auricle and probably due to the auricular contraction while the two others are registered over the ventricles. As regards these two, the former was believed to be due to the distension of the ventricular wall through the flow of blood from the auricle, the other being the result of the transitory and incomplete closure of the auriculo-ventricular valves which takes place after the discontinuation of the blood flow, as reported by HENDERSON and JOHNSON (1912) and DEAN (1916).

The auricular sound which is registered over the precordium is therefore, according to the conception of the majority of investigators, not due to the muscular contraction itself but to vibrations in the ventricular wall or in the auriculo-ventricular valves under the influence of auricular systole. However, an auricular sound can at times be registered even during ventricular systole in cases of complete block, when the auriculo-ventricular valves are closed (MACLEOD, WILSON and BARKER 1931—32). This would indicate that also the auricular contraction is registrable by means of the usual precordial lead.

In accordance with the mechanism of origin, the auricular sound disappears at fibrillation, as pointed out by several authors (MOZER and DUCHOSAL 1930, and others).

At registration the auricular sound is generally found to be separated from the first sound (ORIAS and BRAUN-MENENDEZ 1939, MANNHEIMER 1940) but may, apparently, be included in it in the form of initial vibrations. BRAMWELL (1935 a) observed, in a case of partial block, that the initial vibrations of the first sound in the normal beats began at the same distance from the

TABLE 4.

Frequency of auricular sound in children at phonocardiographic registration.

Author	Age distribution	Number of cases	Percentage frequency of auricular sound
Bridgman (1914)	12—15 years	16	69
Segura (1936)	0—2 »	100	38
Mc Kee (1938 a)	5—17 »	105	94
Mannheimer (1940)	0—14 »	135	54.8±4.3

P wave as the auricular sound in the blocked auricular beats and, further, that the vibrations were identical in both cases. From this he drew the conclusion that the initial vibrations in this case and, probably, also in several others originated from auricular systole and not from ventricular systole. Cossio and Lascales (1936) showed that initial vibrations of this kind in certain instances by means of a sufficiently sensitive apparatus can be registered before the Q wave in the electrocardiogram and, accordingly, must derive from auricular systole in these cases.

The occurrence of the auricular sound.

At exploration via the oesophagus, an auricular sound has, as already stated, been ascertainable in 100 per cent of the examined cases. It is not found equally often with the usual precordial lead. This is, according to Orias and Braun-Menendez (1939), no doubt due to the defective conduction to the surface of the thorax. Braun-Menendez and Orias (1934) were able to register this sound in 20 per cent of 100 healthy males at the age of 20—25 years. Caeirio and Orias (1937) recorded it in 85 per cent of a similar material but using a more sensitive method. Pereira (1939) came across it in 71 per cent of a material of young females. Frost (1944), on the other hand, was able to register an auricular sound in only 2 cases out of 169 with healthy hearts, aged 10—80 years. Thus, the frequency figures presented by different authors are greatly dependent on

the sensitivity of the instrument employed and are, consequently, of minor interest in this connection. They only serve to show that a registrable auricular sound is a normal and frequent phenomenon in young individuals. The physiological auricular sound has also been registered in children in a great number of cases, as may be seen in Table 4.

The gallop rhythm

As previously mentioned, extra sounds may be perceived in healthy individuals, even though they as a rule are not distinctly audible. Extra sounds have also been observed in persons with certain cardiac diseases. The term gallop rhythm has then been used. No proper investigations regarding the extra sounds in healthy individuals, as compared to cardiac patients, have been performed, at any rate not on children. It is conceivable that extra sounds are more common in cardiac cases and the sounds may possibly have a different quality in these. However, the clinical observations of gallop rhythm have generally not been recorded in a way permitting of an estimation of their frequency. Investigations of the sound quality in cardiac patients as compared to healthy individuals, are practically non-existent. The difference between physiological and pathological extra sounds therefore remains to be elucidated. Furthermore, an objective definition of the gallop rhythm is lacking.

The first clear description of the gallop rhythm was presented by CHARCELAY in 1838. However, the term »bruit de galop» was not introduced until 1847 by BOUILLAUD. His follower, POTAIN (1875, 1894, 1900), undertook a very detailed study of the gallop rhythms which has since been of fundamental significance. He described the gallop rhythm as a »triple bruit du coeur constitué par l'addition aux deux temps normaux d'un troisième temps, étranger à ceux-ci, qui n'est ni un souffle ni un frottement mais un bruit frappé, interposé entre les bruits normaux dans l'un ou l'autre silence».

Thus, according to POTAIN, the gallop rhythms could either be systolic or diastolic. Other authors too, have reported cases where extra sounds have appeared between the first and the

second heart sound and, accordingly, defined them as systolic gallops. However, they are rare in comparison with the diastolic forms and have been registered phonocardiographically in but a few cases (EVANS 1943, among others). Their etiology and clinical significance are unknown.

POTAIN divided the diastolic gallop rhythms into two groups according to the localization of the extra sound in diastole, viz., a presystolic and a protodiastolic kind. He also made a distinction between left-sided and right-sided forms, according to whether the gallop was assumed to occur in the left or in the right half of the heart. In the latter case, it was best audible over the inferior part of the sternum or the xiphoid process, while the maximal audibility of the left-sided form was at the apex. POTAIN even drew up a special disease picture, i. e. a »cardiopathie d'origine gastro-hépatique», at which a right-sided gallop rhythm was said to occur. Still, this syndrome has not been confirmed by later investigations (by, for instance, LAUBRY and PEZZI 1926). Other authors have found a right-sided gallop in cases of emphysema and certain chronic lung diseases with hypertrophy of the right half of the heart. LAUBRY, ROUTIER and LARGEAU (1924) considered it an important sign of right-sided cardiac insufficiency. On the whole, the interest in this form of gallop rhythm seems to have waned with the years. However, it was mentioned by WOLFERTH and MARGOLIES in the year 1933. They found it in 5 cases out of 60. Some of these cases suffered from a predominantly right-sided cardiac insufficiency. On the other hand, FROST (1944) did not come across any single case of right-sided gallop rhythm in his material. A right-sided cardiac insufficiency was found in only 1 of his cases and the gallop sound was then best heard over the apex.

The classical form of the gallop rhythm is the left-sided, diastolic one. Since this form has, as a rule, been looked upon as an important clinical sign of a faltering function of the heart, it has been submitted to intensive studies by a great number of investigators. Among the extensive literature on this subject, only a few of the more outstanding works will be dealt with in the present survey. For a more detailed orientation in this respect reference may be had to monographs and surveys pub-

lished by, for instance LAUBRY and PEZZI 1926, HOLT 1927, FROST 1944.

The earliest investigations regarding the gallop rhythm were based only on auscultation and were therefore of rather limited value. The introduction of graphical methods, along with that of auscultation, rendered possible a more accurate analysis of the position of the gallop sounds in the cardiac cycle. The classical registrations which were adopted in this case were the apex-cardiogram and the phlebogram. The apex-cardiogram, with or without simultaneous registration of the venous pulse, is the method principally employed by a great number of authors (CHAUVEAU 1902, MÜLLER 1906, BARIÉ 1912, PEZZI 1914, LAUBRY and PEZZI 1926, and ROUTIER and VAN BOGAERT 1934, among others).

The introduction of phonocardiography together with other methods of registration made it possible to study directly the relationship between the physiological and the acoustic phenomena in the heart and thus to obtain a more definite conception of the mechanism of origin of the gallop rhythms. Synchronous registration of the heart sounds and of the electrocardiogram was performed by among others MOND and OPPENHEIMER 1929, MOZER and DUCHOSAL 1930, DUCHOSAL 1932, 1935, WOLFERTH and MARGOLIES 1933, ROUTIER and VAN HEERSWYNGELS 1935, FROST 1944. ORIAS and BRAUN-MENENDEZ (1939) draw attention to the superiority of the venous pulse curve to the electrocardiogram as a reference curve at phonocardiography, the various phases of diastole not being represented in the latter.

As a rule, the gallop sounds are described as weak and dull, barely audible and accompanied by a palpable throb in the region of the apex of the heart. The gallop rhythm is frequently stated to be easier to feel than to hear. Thus, POTAIN recommended listening with the ear applied directly to the thoracic wall. The intensity of the gallop sound should, according to ROUTIER and VAN BOGAERT (1934 b) be greater in a supine than in an erect position. Also the respiratory phase plays a part in this connection, the gallop rhythm being most distinct at the end of the expirium. The point of maximal audibility is generally stated to lie at the apex or close to it.

Accordingly, the extra sounds in gallop rhythm at auscultation disclose the same properties, broadly speaking, as the physiological third heart sound (and the auricular sound). Furthermore, more recent phonocardiographic works by among others DUCHOSAL (1932), WOLFERTH and MARGOLIES (1933) and BATTRO, BRAUN-MENENDEZ and ORIAS (1934) are considered to have fully elucidated the fact that the gallop rhythm is due to an accentuation of these normal sound phenomena. As a rule, therefore, the earlier, purely descriptive terms presystolic and protodiastolic gallop rhythm are lately being abandoned for the expressions used particularly in Anglo-Saxon literature, viz., auricular gallop or auricular sound gallop and rapid filling gallop (ORIAS and BRAUN-MENENDEZ 1939, FROST 1944), or third sound gallop (MANNHEIMER 1940), respectively. In cases of tachycardia or prolonged conduction time, a summation of these two forms may take place. WOLFERTH and MARGOLIES (1933) have introduced the term summation gallop for these phenomena.

The phonocardiographic methods of registration hitherto employed have not rendered possible a distinction between extra sounds, causing gallop rhythm, and the corresponding physiological sounds. Like the latter sounds, the gallop sounds at registration have been found to consist of but a few vibrations of a comparatively low frequency. However, the registrations have taken place within a certain limited frequency range and no attention has been paid to the occurrence of possible overtones. By means of a method of calibrated phonocardiography MANNHEIMER (1940) showed that the gallop sounds have a higher amplitude as well as a higher frequency than the corresponding normal sounds.

The relationship of the gallop rhythm to the apex-cardiogram, the phlebogram and the electrocardiogram.

The classical registrations used in the study of the gallop rhythms are, as previously mentioned, that of the apex-cardiogram and the phlebogram. In cases of presystolic gallop, an enlargement of the auricular wave (the *a* wave) which has been considered to prove the auricular origin of the gallop sound

has been observed in these tracings (e. g., PEZZI 1914, ROUTIER and VAN BOGAERT 1934 a). In cases of protodiastolic gallop rhythm, the diastolic wave in the apex-cardiogram is said to be enlarged (MÜLLER 1906, PEZZI 1914) but the extra sound is not represented by a wave in the phlebogram.

In later years, the electrocardiograms and the phonocardiograms have to a large extent been registered synchronously. At auricular sound gallop, the extra sound will invariably occur after the P wave in the electrocardiogram. At rapid filling gallop, it will as a rule appear before the P wave but may also at times, in cases of tachycardia, come to have a presystolic position owing to a shortening of the diastole.

The etiology of the gallop rhythm.

The presystolic gallop rhythm has, as a rule, been connected with auricular systole. According to some authors, it is due to an increased auricular contraction on account of hypertrophy (EXCHAQUET 1875, KRIEGE and SCHMALL 1891, ROBINSON 1908, VON WYSS 1910, among others). Autoptic examination has, in several instances though far from always, revealed an auricular hypertrophy.

Other authors regarded a prolongation of the conduction time as an important prerequisite for the occurrence of an audible auricular sound. FOGELSON (1931) assumed the gallop rhythm to be due to a splitting up of the first heart sound into its normal auricular and ventricular parts, either by a prolongation of the P—Q interval or owing to bundle branch block. Later experiences in this matter have shown that a prolonged P—Q may, it is true, at times occur together with the gallop rhythm but that there is no causal connection between the two phenomena (ROUTIER and VAN BOGAERT 1934 c, ORIAS and BRAUN-MENENDEZ 1939).

According to POTAIN, a change in the elasticity of the ventricular wall is the main cause of the gallop rhythm. He believed the extra sound to originate in the structurally altered ventricular wall, when this is suddenly dilated by the inrush of blood from the auricle. The part played by the ventricle in the

occurrence of the presystolic gallop rhythm has also been pointed out by several other investigators (Barié 1912, Laubry and Mougeot 1921, Mozer and Duchosal 1930, among others). Still, in agreement with Potain, these authors do not, as a rule, seem to have attributed any particular significance to the auricular enlargement.

The protodiastolic gallop rhythm was interpreted by the majority of earlier scientists merely as a variation of the presystolic one, where the extra sound has come to lie closer to the preceding second sound than to the following first sound owing to tachycardia or prolonged conduction time. Later investigations, principally with a synchronous registration of the electrocardiogram and the phonocardiogram, have, however, disclosed the fact that the majority of protodiastolic gallop rhythms are not produced by the auricular contraction but constitute rapid filling gallops.

As in the case of the physiological third sound, the extra sound at rapid filling gallop is considered to originate from vibrations in the ventricular wall when this is exposed to a sudden distension by the rapid inrush of blood at the beginning of diastole. The pathologically increased intensity at gallop rhythm is, apparently, due to a reduction in myocardial tone (Orias and Braun-Menendez 1939, and others). A rapid filling gallop would thus, above all, be a sign of myocardial damage.

In contrast to the extensive clinical observations, the experimental investigations regarding the gallop rhythm are extremely rare. Nyboer, Spence and MacNeal (1941) studied the heart sounds and the murmurs in cases of experimentally produced endocarditis lenta on rabbits. 29 animals were examined repeatedly by auscultation. In addition, some of them were subjected to stethography. A gallop sound or »modified» sound in presystole was registered in 4 cases; one of these at autopsy disclosed an extensive myocardial infarction of the left ventricle but no vegetative formations. In another of these cases a distinct murmur developed later as well as a mitral stenosis. However, the extra sounds seem to have been secondary findings at the examination and their significance was not discussed. Nor was any note made of whether a graphical

registration of the heart sounds had also been made before the occurrence of the endocarditis.

LUISADA and MAUTNER (1943) studied the heart sounds and murmurs in animals, above all in dogs, after vagal stimulation, injection of various drugs, etc., by means of simultaneous registration of electrocardiograms and phonocardiograms. Extra sounds, causing a gallop rhythm, occurred under certain experimental circumstances when the vagus stimulation was accompanied by an increased contractility of the myocardium (e. g., by the administration of digitalis, epinephrine). The limit between the gallop rhythm and the corresponding physiological sound seems to have been set in a rather arbitrary fashion by the fore-mentioned authors who were only guided by the amplitudes in a certain frequency range. When these amplitudes were conspicuously high, the definition gallop rhythm was considered justified. Still, no explanation is given of the extent to which the normal variation range has been taken into account in this connection. With this reservation, the conclusion may be drawn from LUISADA's and MAUTNER's investigation that a gallop rhythm may also occur through functional alterations of the myocardium, e. g., by vegetative-nervous means.

The occurrence and clinical significance of the gallop rhythm.

POTAIN and his followers observed the gallop rhythm above all in cases of chronic interstitial nephritis, with hypertrophy and dilatation of the heart, and in arteriosclerosis and aortic defects. SCHRUMPF (1918) used the term »Nierengalopp» for the typical presystolic gallop. The hypertensive-arteriosclerotic diseases play a prominent part along with the coronary affections also in the more extensive gallop materials presented in more recent times (WHITE 1928, MACLEOD, WILSON and BARKER 1931—32, DUCHOSAL 1932, 1935, BRAMWELL 1935 b, HAMILTON 1941, FROST 1944, among others). Of these authors FROST was the only one to make a detailed comparative study in order to determine the frequency with which gallop rhythm occurs in different disease groups and in healthy individuals.

A stethoscopically audible gallop sound was perceived in

FROST's material in 44 per cent in thrombosis of the coronary arteries, in 17 per cent in other arteriosclerotic and hypertensive diseases, and in 15 per cent in aortic defects. On the other hand, the gallop sound was not ascertainable in any single case among the healthy individuals in the corresponding age-groups. FROST therefore attributed the extra sound to pathological circumstances. Further, he stated that the gallop rhythm is rare in cases of mitral stenosis, this opinion, apparently, being shared by the majority of authors with the exception of MANNHEMIER (1942), among others.

A gallop rhythm in connection with acute myocarditis has been ascertained by several investigators and ascribed a varying degree of significance. FRAENTZEL (1881) often came across it in cases of pneumonia and typhoid fever as a sign of serious weakness of the heart, in the latter case usually in connection with an intestinal haemorrhage or profuse diarrhoeas. However, autopsy revealed no pathological changes in the heart. LEYDEN (1882) emphasized the importance of the gallop rhythm as a sign of a dilatation of the left ventricle at diphtheria. According to VIERORDT (1888), in infectious diseases, such as diphtheria, it indicated a serious and often fatal weakness of the heart, above all in children. BLACHER (1914) frequently heard a protodiastolic gallop in cases of severe diphtheria with dilatation and decompensation of the heart. MÜLLER (1906) on the other hand did not consider the prognostic significance of the gallop rhythm in, for instance, typhoid fever to be so grave as many would imply.

It may be questioned whether actually a gallop rhythm has been heard in all these cases of acute infectious disease. Most of these patients were, of course, children and young individuals in whom an audible third heart sound may be a normal occurrence, according to what has now been established. In recent years, the gallop rhythm has been mentioned more rarely in connection with acute infectious diseases. This is, no doubt, partly due to the fact that these cases nowadays are to a large extent treated at special hospitals (epidemic hospitals, children's hospitals) and have, accordingly, generally not been included in the materials published regarding the gallop rhythm. Moreover, a certain cautiousness is probably observed nowadays in

diagnosing gallop rhythm, owing to our present knowledge of the physiological extra sounds. However, FRIEDLÄNDER and MANNHEIMER (1944) and FRIEDLÄNDER (1945) perceived the gallop rhythm in a great number of cases of children suffering from acute rheumatic myocarditis.

Among other diseases where gallop rhythm has been found, the following may be mentioned: chronic adhesive pericarditis (WARBURG 1939, FROST 1944) beriberi (WEISS 1928), pernicious anemia, leucemia and haemorrhagic shock (DUCHOSAL 1932), anemia and hyperthyreoidism (WOLFERTH and MARGOLIES 1940, FROST 1944).

There is a difference of opinion regarding the form of gallop rhythm which is the most common. According to WHITE (1928), the protodiastolic form is the most frequent. However, his investigation was based merely on auscultation. The presystolic gallop is usually considered to be the most prevalent (MACLEOD, WILSON and BARKER 1931—32, DUCHOSAL 1935, FROST 1944). In fact, both BRAMWELL (1935 b) and O'FARELL (1939), only take the presystolic form into consideration in this respect. Finally, WOLFERTH and MARGOLIES (1933) and BATTRO, BRAUN-MENENDEZ and ORIAS (1934) state the summation gallop to be the most common. The former noticed, moreover, that the protodiastolic and presystolic gallop had a tendency to occur simultaneously.

As a rule, the gallop rhythm is considered to be of a serious prognostic significance. However, FROST (1944) seems to be the only one to have compared cases with and without gallop rhythm in one and the same disease group from this point of view. He found that the gallop rhythm has no significance with regard to mortality at a given functional capacity of the heart. On the other hand, it is a valuable sign of low functional capacity and accordingly indicates an unfavourable prognosis. Furthermore, FROST states that the prognosis of the protodiastolic gallop rhythm is inferior to that of the presystolic gallop in the disease group of sclerosis arteriae coronariae-hypertensio arterialis. Also WHITE (1928) considers the protodiastolic form to be the most serious one. Otherwise, the presystolic gallop is generally believed to involve a less favourable prognosis (DUCHOSAL 1935, WOLFERTH and MARGOLIES 1940).

Among 19 cases of gallop rhythm in acute rheumatic carditis in children, FRIEDLÄNDER and MANNHEIMER (1944) detected a third sound gallop in 7, an auricular sound gallop in 5 and a summation gallop in 7. In their opinion, the auricular sound gallop and often also the summation gallop originates from a dilatation of the auricles and, therefore, has a more serious prognostic significance, while the third sound gallop would be benign from a clinical point of view. The number of observed cases in their material, however, is apparently too small to permit the drawing of any definite conclusions.

Finally, an investigation by FAZEKAS (1942 a) will be mentioned in this connection. FAZEKAS perceived a gallop rhythm in 108 cases out of 1300 school children at the age of 7—20 years, 50 being boys and 58 girls. However, the author seems not to have made a distinction between the gallop sound and the audible physiological third sound. 8 of the boys were ≤ 12 years and $42 \geq 13$ years. Of the girls, 8 were ≤ 10 years and $32 \geq 14$ years. Thus, FAZEKAS considers the gallop rhythm to be a rare phenomenon before the age of puberty but fairly common during adolescence. 41 of the boys and 27 of the girls regularly joined in the athletic sports and practically all the rest took part in gymnastics. This would indicate that the gallop rhythm is more common among school children practising athletics than among those not participating. However, the number of children doing athletic exercises among those not manifesting any gallop sounds had not been specified. FAZEKAS (1942 b) also examined a small number of young men performing heavy work (blacksmith's apprentices), some of whom in addition regularly took part in athletic sports. FAZEKAS found that the frequency of gallop rhythm among those performing only manual labour was not greater than that of school children of the same age, but that heavy manual labour combined with regular physical exercises greatly added to the prerequisites of gallop rhythm. According to the same author, this gallop sound would probably most often be a manifestation of increased vagal tone but, nevertheless, a sign which should induce extra caution.

Changes in the electrocardiogram at gallop rhythm.

The majority of examinations regarding the gallop rhythm have been performed on elderly people with clinical symptoms of cardiac disease. It is therefore natural that pathological changes have often been observed in the electrocardiogram in cases of gallop rhythm.

Tachycardia thus seems to be the rule and, according to some authors such as, for instance, BRAMWELL (1935 b) constitute a conditio sine qua non. WHITE (1928) ascertained in his material a heart frequency of, on an average, 100 beats per minute, having a pulse frequency of between 80 and 120. 28 of HAMILTON's (1941) 32 cases had a pulse frequency of > 90 beats per minute.

ROUTIER and VAN BOGAERT (1934 a) and DUCHOSAL (1935) often observed an enlarged and widened P wave at auricular sound gallop.

Several authors have often come across a prolongation of the conduction time at gallop rhythm (GALLAVARDIN 1920, BOURDET, YACOËL and GIROUX 1923, and ROUTIER and VAN BOGAERT 1934 a, among others). It has been pointed out earlier that a disturbance of conductivity between auricle and ventricle has been considered by some investigators as an important prerequisite for the occurrence of gallop rhythm, but that later investigations have shown that there is no causal connection between these phenomena. According to WHITE (1928), auriculo-ventricular block is rather a rare occurrence at gallop rhythm. Furthermore, FROST (1944) found a normal P—Q interval in 85 per cent with an auricular sound gallop.

Bundle branch block has also been considered a comparatively common finding at gallop rhythm. WHITE (1928) observed this abnormality in 38 per cent. FOGELSON (1931) is of the opinion that bundle branch block is a prerequisite for the occurrence of gallop rhythm in certain cases. He considered himself to have confirmed his assumption by experiments on dogs. On the other hand, LEWIS (1934) did not believe in any causal connection between the two phenomena. In his opinion the relatively high frequency of gallop rhythm in cases of bundle branch

block (in 9 cases out of 23 in his material) is due to the fact that both these conditions occur at the same types of heart disease.

Apart from the changes in the electrocardiogram mentioned above, a number of other changes have been described by several authors, such as widening of the QRS complexes, notchings, inverted T waves, arrhythmias and extrasystoles (cp. BOURDET, YACOËL and GIROUX 1923, WHITE 1928, ROUTIER and VAN BOGAERT 1934 c, among others). However, the latter did not find any parallelism whatsoever between the electrocardiographical changes and the gallop rhythm.

But a few investigations have been performed on children and young persons. FAZEKAS (1942 a) found, among his 108 school children with gallop rhythm (audible third sound), a prolonged conduction time (P—Q $>$ 0.20 seconds) in 44 per cent of the boys and 24 per cent of the girls, while about half of them had a P—Q interval of $<$ 0.15 seconds. Apart from this only insignificant changes occurred in the electrocardiogram. FAZEKAS believed the disturbed conductivity in these cases to be caused mainly by an increased vagal tone. In FROST's (1944) material of gallop rhythms, a small group of young patients was included who suffered from »potential» heart diseases, usually febris rheumatica peracta. These cases disclosed normal electrocardiograms throughout.

Röntgenological changes of the heart at gallop rhythm.

The left-sided gallop rhythm has for long been connected with a hypertrophy and dilatation of the left ventricle in several instances. This was, as a rule, ascertained by means of palpation and percussion. Lately, Röntgen has also been used. ROUTIER and VAN BOGAERT (1934 b) state that the heart shadow invariably is enlarged in the case of gallop rhythm. HAMILTON (1941) noticed an enlargement in 29 out of 32 cases (only the uncertain cases, however, being subjected to a radiological examination). In studying the hypertensive-arteriosclerotic diseases, FROST (1944) more frequently observed an enlargement of the heart in the cases with a gallop rhythm than

in those without. In the small group of young patients with a gallop rhythm and »potential heart disease» previously mentioned, he was on the other hand unable to find any pathological changes in the Röntgen picture. Not less than 3: of FAZEKAS' (1942) 108 cases of gallop rhythm in school childre (referred to above on page 27) disclosed changes at the Röntge examination. 12 of these had an enlargement of the left ven tricle and 5 of the right one, another 5 having an enlarge left auricle. In addition, 6 of the remaining cases had a bulgin pulmonary arch and 4 a mitral configuration. Apparently, th author made no comparison with the cases without gallo rhythm, and it appears reasonable to believe that the signi ficance of the röntgenological observations has been over estimated. In children subjected to rheumatic carditis, FRIED LÄNDER and MANNHHEIMER (1944) in 9 cases out of 12 displayin an auricular sound gallop or summation gallop ascertained an auricular enlargement in the röntgenogram. Here also a com parison between cases with and without a gallop rhythm is lacking.

Patho-anatomical changes at gallop rhythm.

GARVIN (1943) examined the frequency of gallop rhythm in a section material comprising 790 adult patients who died of a cardiac disease. 199 of these, or 25.2 per cent, had an auricular or third sound gallop. It was most common in hyper tension and coronary affections, the frequency being 1 out of 3, while the frequency in rheumatic and syphilitic cardiac diseases equalled 1 out of 10. The cases with a gallop rhythm, when compared to those without, showed a lower age at death. This figure was statistically significant.

Differential diagnosis.

a) *Duplicated first heart sound.* A distinction was made between gallop rhythm and duplicated heart sounds, already by POTAIN. A »dédoublement» is characterized by the fact that both components have the same quality and are separated from one another by a very short interval. The gallop sound, on

the other hand, has quite a different sound quality than the other heart sounds, being separated from them by a longer interval.

A duplicated first heart sound is best perceived over the apex or between the apex and the xiphoid process, often being most distinct at the end of the expirium. According to LIAN and WELTI (1938), it is most easily heard in an erect position. The graphical registration of this sound shows two groups of vibrations instead of one continuous sound. They are sometimes separated by a distinct interval which, however, does not exceed 0.02—0.03 seconds. At other times this interval is lacking and the first sound is registered by two amplitude maxima. The first group of vibrations begins at the same distance from the Q wave as the undivided first sound, and the total duration of the two groups either does not exceed the duration of this sound or does so but slightly (LIAN and WELTI). The two vibration groups are rarely altogether identical as regards amplitude or frequency.

There is a diversity of opinion regarding the mechanism of origin. Some authors consider the duplicated first sound as an important sign of bundle branch block. However, phonocardiographic investigations by among others COSSIO and BRAUN-MENENDEZ (1935) have disclosed that what is conceived at auscultation as a duplicated first sound is frequently at graphical registration found to be an auricular sound, and presystolic gallop often seems to occur at bundle branch block, in particular (MACLEOD, WILSON and BARKER 1931—32, BATTRO, BRAUN-MENENDEZ and ORIAS 1936, among others). Other authors, such as LIAN and WELTI (1938) find it improbable that an asynchronism in the contraction of the left and the right half of the heart could be so pronounced as to give rise to a duplicated first sound. These investigators instead agree with ORIAS and BRAUN-MENENDEZ, explaining the duplication as an accentuation of a normally occurring, more or less distinct, differentiation of the first heart sound into two parts, which would correspond to the isometric and ejection phases, respectively. Whatever the exact interpretation conceived in this matter, there is nowadays no tendency towards attributing a duplication of the first heart

sound any pathological significance. It has been perceived frequently also in individuals with perfectly healthy hearts.

b) *Duplicated second heart sound.* This sound is best heard at the base, above all in the second intercostal space to the left of the sternum and, as in the case of the duplicated first sound, most distinctly towards the end of the expirium. It is represented graphically by two vibration groups of similar appearance, separated by a short interval not exceeding 0.03 seconds. The first vibration group begins at the same place in relation to the T wave as the continuous (undivided) second sound, but the total duration of the two is greater than that of the second sound (Lian, Minot and Welti 1941). The duplication is due to an asynchronous closure of the aortic and pulmonary valves, a phenomenon occurring quite frequently under normal conditions from what has been established through animal experiments. Accordingly, a duplicated second sound does not necessarily have a pathological significance. Simultaneous registration of the heart sounds and the venous pulse will show that the later phase of the duplicated second sound corresponds to the ascending line of the *v* wave (see Fig. 1), that is to say the moment before the opening of the auriculo-ventricular valves. The third heart sound or the rapid filling gallop on the other hand has a localization in the diastole corresponding to the descending line of the *v* wave, i. e. the rapid filling phase.

c) *Opening snap of the mitral valve.* In mitral stenosis a triple rhythm often occurs, where the extra sound is perceived just after the second heart sound and should rather be called a short snap than a sound. It is often observed over the entire precordium but, at times, only over a limited area between the apex and the left sternal border. It was first described by Duroziez (1862) who, however, gave an erroneous interpretation of this sound as due to an asynchronous closure of the aortic and pulmonary valves. Potain's disciple, Rouches (1888), made a distinction in cases of mitral stenosis between two kinds of triple rhythm. One was best audible over the base and assumed to be caused by an asynchronous closure of the semilunar valves. The other had its maximal point of audibility over apex, the extra sound here being of a different nature to that of the

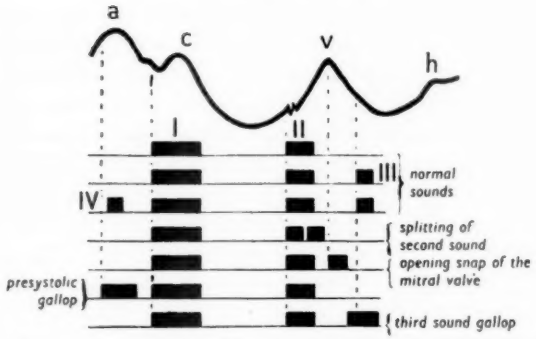


Fig. 1. *Schematic picture of the heart sounds, the gallop sounds and the venous pulse.* According to Cossio and Orias (1935).

The second component of the split second sound coincides with the ascending line of the *v* wave, the opening snap of the mitral valve with the summit of the *v* wave and the third sound with the descending line of the *v* wave. I = first sound, II = second sound, III = third sound, IV = auricular sound.

second sound and separated from it by a longer interval. It was supposed to originate from a tension in the sclerotic mitral valves at their opening at the beginning of diastole and was, therefore, called the »*claquement d'ouverture de la mitrale*». THAYER (1909) used the expression *opening snap of the mitral valve.* ROUCHES' observations have been confirmed by phonocardiographic investigations in later years by among others MARGOLIES and WOLFERTH 1932, LIAN, MINOT and WELTI 1941. Apart from a duplicated second heart sound over the base which is indistinguishable from the physiological one, a triple rhythm is, accordingly, to be found at mitral stenosis which is audible and registrable in over half of the cases and pathognomonic of this disease. The distance of the extra sound from the preceding second heart sound is longer than the interval between the two phases of the duplicated second sound but shorter than the distance of the third heart sound from the second sound. MARGOLIES and WOLFERTH state the distance to equal 0.06—0.11 seconds while LIAN and others give 0.08—0.09 seconds. The opening snap often directly passes over into a diastolic murmur.

Sometimes a short interval between the two is heard and registered. At simultaneous registration of the heart sounds and the venous pulse, the opening snap coincides with the summit of the *v* wave (MARGOLIES and WOLFERTH 1932, COSSIO and ORIAS 1935). This facilitates a differential diagnosis in uncertain cases (see Fig. 1). The localization of the extra sound in relation to the curve of the venous pulse, moreover, confirms the earlier assumption that it occurs at the opening of the sclerotic mitral valves.

d) *Murmurs in mitral stenosis.* The presystolic murmur in mitral stenosis may sometimes be confused with the presystolic gallop and the differential diagnosis may be impossible without the help of phonocardiography. WOLFERTH and MARGOLIES (1933) had several cases of presystolic gallop in their material which were erroneously interpreted as presystolic murmurs at auscultation. MANNHEIMER (1941) draws attention to the relationship between the auricular sound and the presystolic murmur, stating that both occur in mitral stenosis with auricular enlargement and that the murmur may often be defined as overtones of a pathologically accentuated auricular sound. Similarly, MANNHEIMER points out that a protodiastolic murmur may at times form the overtones of a rapid filling gallop. A short diastolic murmur may at auscultation be perceived as a third sound. The phonocardiographic registration is therefore of decisive significance with regard to the differential diagnosis. However, according to MC KEE (1938 b) it is not always easy to determine even on the phonocardiogram whether the vibrations registered at the beginning of diastole constitute a sound or a murmur.

e) *The physiological third heart sound and auricular sound.* While the normal auricular sound is practically inaudible and therefore hardly gives rise to any complications in the differential diagnosis at auscultation, the physiological third heart sound has often been mistaken for a gallop rhythm. This applies above all to children and young individuals where the forementioned sound is perceptible under normal circumstances. GUBERGRITZ (1929) states that the third heart sound is distinguishable from the protodiastolic gallop by a different

acoustic quality and intensity. On the other hand, MELIK-GÜLNASARIAN (1932) was unable to find any definite difference in the intensity and he maintained that a palpable throb could be perceived in both cases. LIAN and RACINE (1933) could not differentiate between a gallop and the third heart sound at auscultation and the same opinion was propounded by ROUTIER and VAN BOGAERT (1934 b).

Even by means of the phonocardiographic registration methods hitherto employed it has not been possible to distinguish between the pathological and the physiological extra sounds (LIAN and RACINE 1933, WOLFERTH and MARGOLIES 1933, CALO 1938, FROST 1944). Owing to the fact that the latter are registered to a far greater extent than they are audible at mere auscultation, which applies especially to the auricular sound, phonocardiography in its present form has, on the contrary, further increased the confusion in this field. The gallop rhythm has been diagnosed in far too many cases where it undoubtedly was only a question of a normal third sound or auricular sound.

However, by means of calibrated phonocardiography it should, according to MANNHEIMER (1940), be possible to obtain a phonocardiographic definition of the gallop rhythm which would make the physiological and the pathological extra sounds distinguishable from each other.

Summary

The extra sounds in healthy individuals are of two different kinds, viz., a third heart sound and an auricular sound. The third sound is due to vibrations in the ventricular wall probably owing to the rapid inrush of blood into the ventricle at the beginning of diastole. The auricular sound occurs in connection with auricular systole, several factors in all likelihood cooperating in this case. Both are frequently registered phonocardiographically, above all in children and young individuals. However, the auricular sound is practically inaudible under normal conditions while the third sound has been perceived by different authors to a varying degree.

Extra sounds are also observed in certain cardiac diseases, and the term gallop rhythm is then generally used. There are two different kinds of gallop rhythm, viz., the third sound gallop (or rapid filling gallop) and the auricular sound gallop. The extra sounds at gallop rhythm are identical in several respects with the normal third sound and auricular sound, respectively, and are considered to be an accentuation of these sounds. The third sound gallop is believed to originate from a pathologically reduced tone of the ventricular wall, although experimental proof of this is lacking. The auricular sound gallop is probably also due in part to such a decrease in tone to which may perhaps be added an increased auricular contraction.

The gallop sounds have been submitted to detailed analysis in elderly persons, among whom they seem to appear chiefly in hypertensive-arteriosclerotic and coronary diseases, and have been attributed to failing cardiac function. Since extra sounds are practically non-existent in healthy individuals in the corresponding age-groups, there is reason to assume that they are pathological in this connection.

The gallop rhythm is also said to occur among children and young people and, above all, in acute myocarditis. However, the extra sounds in these cases cannot be regarded as definitely pathological, since they are a normal occurrence in the age-groups concerned here. It is conceivable that the extra sounds are more common in cardiac disease and the sounds may, possibly, have a different quality. Still, no investigations have been performed proving this. As a rule, the pathological and physiological extra sounds are considered to be indistinguishable from one another whether at auscultation or by means of the phonocardiographic registration methods hitherto employed. According to the majority of authors, the only criterion of a pathological extra sound is the fact that other signs of cardiac disease are present simultaneously. On the other hand, when the cardiac function is normal, the extra sound is regarded as physiological.

However, by the use of the forementioned definition, we are deprived of the possibility of utilizing the gallop rhythm as a diagnostic aid. It is, therefore, necessary to try to arrive at

a definition of the gallop rhythm which is based on an objective method. This has been rendered possible by means of calibrated phonocardiography, as demonstrated earlier by MANNHEIMER (1940).

Thanks to this method, the absolute amplitude of the heart sounds can be measured in dynes per cm^2 within the various frequency ranges (cp. the chapter on Methods). By determining the normal variation range of the third heart sound and the auricular sound, an upper limit can be established with regard to the amplitude which may not be exceeded if the sound in question is to be regarded as physiological.

The purpose of the present work is in the first place to offer a phonocardiographic definition of the gallop rhythm, based on calculations made on a normal material of children.

By means of a clinical investigation, attempts will then be made to derive information regarding the significance of the gallop rhythm in childhood.

An experimental study of the gallop rhythm has been made, with a view to shedding further light on the problem.

CHAPTER II

Methods

Apparatus

The phonocardiographic registration has been performed by means of the apparatus constructed by MANNHEIMER and STORDAL and described by them in detail earlier (MANNHEIMER 1940). A brief recapitulation of their account of this apparatus may be appropriate in this connection.

From a crystal microphone with closed chamber, the impulses are directed to the phonocardiograph. They are then divided

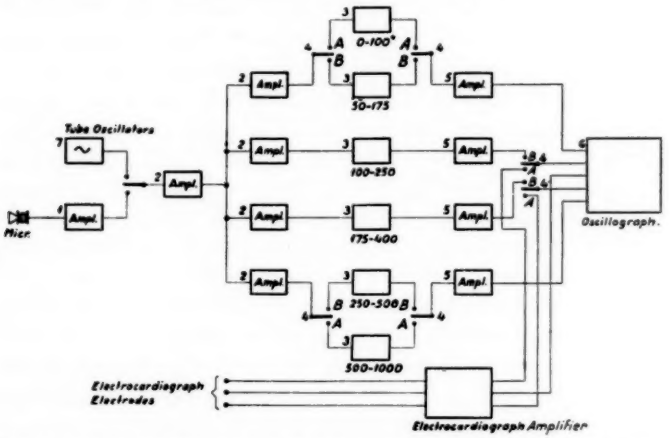


Fig. 2. *Schematic diagram of the phonocardiograph* (MANNHEIMER, 1940). 1 = the input of the apparatus. 2 = pre-amplifier. 3 = filters. 4 = switching arrangements. 5 = post-amplifier. 6 = electrocardiograph with built-in oscillograph coils. 7 = tube oscillators.

A — alternative A (2 phonocardiograms, 3 electrocardiograms) } see text
B — alternative B (4 phonocardiograms, 1 electrocardiogram) } page 40.

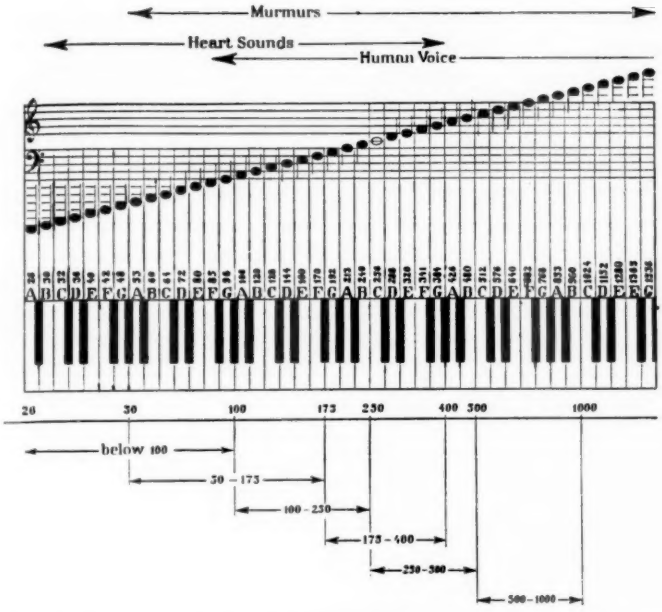


Fig. 3. The position of the six frequency ranges in the tonal scale (according to MANNHEIMER, 1940).

into four channels, containing four uniform amplifiers functioning independently of one another. From there the impulses are led into a filter-system (Fig. 2: 3).

The apparatus contains two types of filter, viz., high-pass filters obstructing the low frequencies but admitting the high ones, and low-pass filters operating in the opposite way. These filters have been rendered strongly selective so as to divide the whole tonal scale covered by the sound phenomena of the heart into a series of separate frequency bands which overlap one another (Fig. 3).

By means of a switching arrangement (Fig. 2: 4), two of the four above-mentioned channels can be given different frequency ranges, alternately.

From the filter units the impulses are led to the post-

amplifiers and from there to the oscillographs which are built into the electrocardiograph (Fig. 2: 6) which is of a three-lead type (the Elmqvist system).

By means of the forementioned switching arrangement, all three electrocardiographic leads and two frequency ranges in the phonocardiogram can be registered simultaneously in the one case (alternative A) and in the other only lead II in the electrocardiogram and the remaining four frequency ranges (alternative B). Thus, for every patient two recordings are obtained in accordance with the following scheme (PCG = phonocardiogram, ECG = electrocardiogram).

A	PCG	below 100 cycles per second
	ECG	lead I
	ECG	lead II
	ECG	lead III
	PCG	500—1000 cycles per second
B	PCG	50—175 cycles per second
	PCG	100—250 cycles per second
	ECG	lead II
	PCG	175—400 cycles per second
	PCG	250—500 cycles per second

Two different rates of film speed are used, i. e. one of 40 and another of 100 mm per second. The time is recorded every 0.02 seconds.

In addition to the amplifier and filter appliances described above, a built-in contrivance for calibration of the amplitude of the in-coming sound phenomena is to be found in the apparatus. It consists of tube oscillators emitting sounds of known frequency and amplitude. By means of voltage dividers, consisting of a tapped resistance, the full voltage sent out in the tube oscillators can be reduced. Thus, the voltage of the impulses coming from the microphone can be compared in each single case. This voltage is measured in millivolts.

The crystal microphones employed are not equally sensitive in spite of their uniform type. Accordingly, attempts were made to arrive at absolute measures of the sound amplitude. This

was, in fact, achieved by means of calibration of the microphone before use in a special microphone calibrator. This way, the microphone is exposed to known pressure variations and the voltage can be measured. The amplitude of the sound phenomena is expressed in units of pressure, i. e. in dynes per cm^2. A reproducible method has thus been obtained by means of which a comparison of the results found with different microphones and different apparatuses can be performed.

This method of calibrated phonocardiography, as elaborated by MANNHEIMER and STORDAL, offers possibilities of quantitative measurement of the amplitudes of heart sounds and murmurs within different frequency ranges, as well as of comparison between these sound phenomena in normal and pathological conditions.

Statistical methods

Current statistical methods have been employed in the statistical analysis, the standard deviation σ being calculated according to the following formula:

$$\sigma = \pm \sqrt{\frac{\Sigma a^2}{n-1}}$$

where a indicates the deviation from the mean and n the number of observations. When n exceeds 50 the following formula has been used:

$$\sigma = \pm \sqrt{\frac{\Sigma a^2}{n}}$$

The standard error of the mean (M) is computed according to the formula:

$$\varepsilon(M) = \pm \frac{\sigma}{\sqrt{n}}$$

The standard error of a percentage (p) is obtained by means of the formula:

$$\varepsilon(p) = \pm \sqrt{\frac{p(100-p)}{n}}$$

where p is the percentage and n the number of observations.

The standard error of a difference (D) is calculated according to the formula:

$$\varepsilon(D) = \pm\sqrt{m_1^2 + m_2^2}$$

where m_1 and m_2 indicate the standard errors of the means in question.

BRAVAIS-PEARSON's correlation coefficient and its standard error are calculated in the usual manner.

CHAPTER III

Experimentally produced gallop rhythm

As already pointed out, gallop rhythm in human beings has, as a rule, been attributed to myocardial damage. The precise mechanism of origin is, however, far from elucidated, nor have any experimental investigations been performed, as far as is known, in order to prove such a connection. Therefore, a detailed analysis of these conditions in animals seems justified. The method originally described by FLEISHER and LOEB in 1909 seemed the most suitable for producing myocardial damage (see below). The principal features in the following account have been presented before in a preliminary publication (CARLGREN 1945).

Earlier investigations regarding myocarditis after coffein-adrenalin injections

Numerous animal experiments have been performed in order to study the myocardial injuries. These injuries have been produced in different ways, inter alia, by the injection of bacteria, toxins and drugs. The particular method selected here was the one originated by FLEISHER and LOEB (1909). These authors gave intravenous injections of 1.2 cg spartein or 2.5 cg coffein per Kg of body weight to rabbits. Two minutes later 0.2 mg. adrenalin was injected intravenously. This way they obtained macroscopic lesions in the left ventricle in 60 per cent of the animals, practically always located close to the base of the heart. The injured areas contrasted greatly with the surrounding muscle. Their colour was a pale, yellow-brown and their consistency was firm and inelastic. The changes were observed

as early as on the 4th day after the injection, being most distinct from the 10th day onwards. A dilatation of the heart, mainly of the right ventricle, was noticed in a few cases. A hypertrophy was often found. Microscopic changes were ascertained in a great number of cases, examined from 7 seconds up to 6 weeks after the injection. They consisted of degeneration and hypertrophy of the muscle fibres, as well as of an increase of connective tissue.

CHRISTIAN, SMITH and WALKER (1911) used the same drugs but compared the effect of various administration methods and of different doses. They obtained the most marked changes after intravenous injection of large doses of spartein and adrenalin.

BECKE, JOHNSON and HARRIS (1926) studied the changes in the electrocardiogram after coffein-adrenalin injections. Changes of this kind occurred in 12 out of 13 test animals, consisting of inverted T waves of a more or less transient nature. No abnormalities were observed as regards the P waves, the P—R interval or the QRS complexes. There was no absolute parallelism between the electrocardiographic and the patho-anatomical changes.

BARKESDALE (1927) studied the mechanism of origin of the myocardial lesions, using FLEISHER and LOEB's method. He tried to prevent an increase in the blood pressure by simultaneously injecting adrenalin and amyl-nitrite. He thereby succeeded in avoiding myocardial damage in a great number of cases. According to BARKESDALE, this would indicate that the changes are greatly due to mechanical conditions connected with the increase in blood pressure.

JOHNSON and SIEBERT (1927—28) on the whole confirmed FLEISHER and LOEB's results but, in addition, offered more careful statistics regarding the localization of myocardial lesions. The latter were found in almost 100 per cent of the cases and most often in the papillary muscles. They were next observed in the upper half of the anterior ventricular wall, the left auricle and the upper half of the posterior ventricular wall. No lesions were ascertained in the right half of the heart. As regards the microscopic changes, the absence of a leucocytary reaction in the myocardium was reported, amongst other things. The electro-

cardiographic examination revealed pathological changes in all cases.

WASSERMEYER and ROHRBACH (1932) examined the colloid-chemical processes in myocarditis after coffein-adrenalin injections. Contrary to conditions in experimentally produced malformations, they did not, in these cases, find any symptoms of over-fatigue in the myocardium, nor hypertrophy. The heart was often found to be very strongly dilated, a fact that had also been established by means of Röntgen. The increase in size was particularly marked to the right, but the auricles were also affected. The dilatation was as a rule ascertained on the 5th or 6th day after the injection.

GRUBER, OLCH and BLADES (1933) analysed the effect of a number of other drugs as well as the effect on the blood pressure. Myocarditis could be produced by the intravenous injection of any of the drugs belonging to the methyl-xanthin group, provided that this was followed, within a few minutes, by a suitable dose of adrenalin. According to these authors, several factors influence the occurrence of cardiac changes, viz., (a) an increase in the blood pressure, (b) a vaso-constriction of the coronary vessels, (c) a reduction in the blood pressure, leading to malnutrition of the myocardium and (d) a toxic effect of the drug itself.

Further, DECHARD, HERRMAN and ERHARD (1936) followed the creatinine content in the myocardium from a few hours up to one month after the coffein-adrenalin injection. This decreased in relation to the amount of myocardial lesion increasing again by degrees to normal levels, parallel to the development of the reparative process.

Thus, a very high percentage of myocardial lesions have been producible by means of FLEISHER and LOEB's method. They have, as may be seen above, been studied in detail, inter alia, with regard to the localization, the macro- and microscopic appearance, the occurrence of hypertrophy and dilatation, as well as electrocardiographic changes. On the other hand, no corresponding phonocardiographic examination has been performed.

Material and methods

The material comprises 41 young, practically fully grown rabbits with a body weight varying between 2.1 and 4.3 Kg. After a combined electrocardiographic and phonocardiographic examination, 2.5 cg coffein per Kg of body weight were injected intravenously and after another 2—3 minutes 0.2 mg adrenalin. 11 of the animals died directly owing to the injection. The 30 surviving animals were then subjected to repeated electro- and phonocardiographic examinations with, to begin with, shorter and, later, longer intervals. Röntgenograms of the hearts of 24 animals were taken before as well as several times after the injection and, as a rule, simultaneously with the forementioned examinations. One of the rabbits died spontaneously after 11 days and another two after 10 days and 6 weeks, respectively, during attempts to reduce the frequency of the pulse by means of acetylcholine. The rest were killed after 2—8 weeks. By mistake one of the animals was not subjected to an autoptic examination. Otherwise, the hearts of all these cases were examined macro- as well as microscopically.

At the electro- and phonocardiographic examination, the animals were tied down in a supine position and a crystal microphone with a closed chamber was applied over the precordium. Ordinary injection needles were used as electrodes which were introduced subcutaneously into both the forelegs and the left hind leg. Apart from this, the same apparatus was employed as described earlier (cp. the chapter on Methods).

The Röntgen examination was performed in a supine as well as in a lateral position, with a constant distance between the focus and the film. The size of the cardiac shadow was determined by measuring the maximal breadth of the frontal picture and the sagittal diameter, respectively. The degree of enlargement was in each case estimated by comparing the size of the heart after the injection to the original values, and the varying size of the thorax of the animals need therefore not be taken into consideration. Generally, only one examination was performed before the injection. However, occasionally two determinations were made with an interval of a few days, the differ-

TABLE 5.

Normal variation of heart size in Röntgen picture at one and the same examination in one case.

Frontal diameter		Sagittal diameter	
Breadth in mm	Number of determinations	Breadth in mm	Number of determinations
28	2	25	2
29	4	26	6
30	8	27	5
31	1	28	2

ence in size never exceeding 2—3 mm as regards either of the frontal and the sagittal diameters.

In order to obtain an indication of the extent to which the size of the cardiac shadow may vary normally, at a single examination, owing to different phases of contraction of the heart and respiration, 15 determinations were made in one case in a supine and lateral position. The result is shown in Table 5.

Thus, the size of the heart was found to vary normally by at least 4 mm as regards either of the frontal and sagittal diameters. If the figure obtained by adding the increase in size of the frontal diameter to that of the sagittal diameter exceeded 10 mm, this was regarded as a definite sign of enlargement.

In estimating the patho-anatomical changes the hearts of 10 healthy animals not previously treated were, for the sake of comparison, examined macro- as well as microscopically.

The normal phonocardiogram in rabbits

The examination of the 41 rabbits not previously treated gave the following results:

The first and second heart sound was almost always registered up to 500 cycles per second (see the chapter on Methods). The first sound as a rule disclosed a larger amplitude than the second sound, above all in the lower frequency ranges. Only the first sound was generally distinctly perceived at auscultation.

A faint *extra sound* was registered in 2 cases at up to 175 cycles per second after the P wave in the electrocardiogram. A pronounced sinus arrhythmia occurred in one of these cases. Owing to the fact that the distance of the extra sound from the second sound varied between 0.16 and 0.22 seconds, while the distance to the subsequent first sound constantly equalled 0.04 seconds, this extra sound could be defined as an auricular sound. In the other case, it was impossible to determine whether the extra sound was a third heart sound or an auricular one owing to tachycardia. The amplitude was but slight in both cases and considerably below that of the first and second sound.

Faint *systolic murmurs* were registered in over half of the cases in the frequency range of 50—175 cycles per second, becoming gradually less common in the higher frequency ranges (Table 6). Thus, as in human beings the systolic murmur is a normal occurrence in rabbits (Mc Kee 1938 a, Mannheimer 1940).

No *diastolic murmur* was perceived in the animals not submitted for treatment.

Changes in the hearts of rabbits after coffein-adrenalin injection

The electrocardiogram:

An inverted T_1 occurred in 19 cases (see Figs. 6 b—c and 9 b—c) and an inverted T_2 in 8 cases out of the 30 animals surviving the injection. In 7 of these cases, the T wave was simultaneously inverted in leads I and II.

Levine (1942) found an inverted T_1 in 2 out of 23 healthy rabbits and, consequently, did not regard this as a pathological sign. However, he never subjected the animals to a patho-anatomical examination. Therefore, the possibility of a »spontaneous» myocardial lesion in these cases cannot be entirely excluded. Nyboer (1940) described a case of spontaneous changes in the S—T segments of a rabbit where the microscopical examination revealed myocarditis. Furthermore, the author of the present work has on one occasion seen an inverted T_1 appear spontaneously in a rabbit. In this particular instance, a gallop rhythm set in at the same time, as well as a slight enlargement in the Röntgen picture. In addition, the patho-

TABLE 6.

Measurable amplitudes of heart sounds and murmurs in different frequency ranges in normal rabbits.

n_1 = number of cases including 0 figures.
n_2 = » » » excluding 0 figures.

Frequency range cycles per sec.	n_1	n_2			
		First sound	Second sound	Extra sound	Systolic murmur
< 100	41	41	41	2	.
50–175	41	41	41	2	24
100–250	41	41	41	0	14
175–400	41	41	40	0	6
250–500	41	39	40	0	1

anatomical examination disclosed a slight, diffuse increase of the interstitial connective tissue in the myocardium and a certain hypertrophy of the muscle fibres. This was, in all likelihood, also a case of »spontaneous» myocardial lesion.

In the cases not subjected to treatment, T_1 and T_2 were almost invariably positive, in exceptional cases isoelectric but never inverted. Accordingly, an inversion of T_1 as well as T_2 should be judged as pathological.

After the injection, in 1 of the animals a distinct elevation of the S—T segments occurred in lead II (> 1 mm) simultaneously with a change in the direction of the electrical axis from $+ 50^\circ$ to $- 35^\circ$.

In 2 cases, extra systoles were noticed in combination with inverted T_1 and T_2 and, in another 3 cases, the direction of the electrical axis deviated considerably after the injection ($- 5^\circ \rightarrow - 70^\circ$, $+ 30^\circ \rightarrow - 110^\circ$ and $+ 35^\circ \rightarrow + 160^\circ$).

Thus, 21 of the 30 rabbits surviving the injection had pathologically changed electrocardiograms. No changes were ascertainable in the 9 remaining cases, or they were too inconspicuous to be attributed any definite pathological significance, i. e. in one case T_1 was diphasic on only one occasion, and in another case a slight elevation of the S—T interval was perceived, though it did not exceed 0.5 mm.

TABLE 7.

Measurable amplitudes of heart sounds and murmurs in different frequency ranges in rabbits after coffein-adrenalin injection.

n_1 = number of cases including 0 figures.
n_2 = » » » excluding 0 figures.

Frequency range cycles per sec.	n_1	n_2			
		First sound	Second sound	Extra sound	Systolic murmur
< 100	30	30	30	16	·
50—175	30	30	30	16	20
100—250	30	30	30	12	18
175—400	30	30	30	9	10
250—500	30	30	30	6	5

The electrocardiographic changes, which were in good conformity with those observed by BECKE, JOHNSON and HARRIS (1926), occurred during one of the first 10 days after the injection, usually on the 4th to the 6th day. These changes disappeared in a good half of the cases within a space of time varying from a few days to several weeks, while they remained during the whole of the observation period in the rest of the cases, i. e. as a rule for 5—8 weeks.

The phonocardiogram:

No definite changes were observed as regards the *first and second heart sounds.* They were, as in the case of the animals not subjected to treatment, without exception registered up to 500 cycles per second, and the first sound showed a generally larger amplitude than the second sound, above all in the lower frequency ranges.

Systolic murmurs of a high frequency were more often met with here than among the animals not subjected to treatment (cp. Tables 6 and 7). No *diastolic murmurs* were registered.

An *extra heart sound* was observed in 16 of the 30 rabbits who survived the injection (see Table 7). In 12 of these cases, it attained the frequency range of 100—250 cycles per second or higher, the amplitude in the two or three lowest frequency

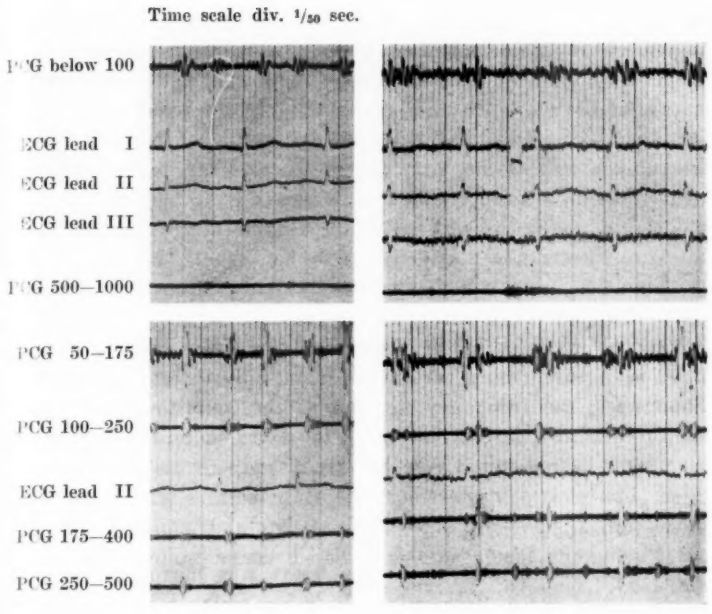


Fig. 4 a and b. Rabbit 6. a) ECG and PCG before injection: Only the two normal heart sounds are recorded. — b) ECG and PCG 6 days after coffein-adrenalin injection: Gallop rhythm. The extra sound registered up to the frequency range of 250—500 cycles per second. The amplitude in the lower frequency ranges is larger than that of the first and second sound. No definitely pathological changes in the ECG.

ranges, as a rule, exceeding that of the second and often even that of the first sound (see Figs. 4 b, 6 b and 9 b). The extra sound differed in these cases, both as regards frequency and amplitude, from the extra sounds occasionally found in the animals not subjected to treatment. It could therefore be considered a pathological sign. According to the definition made on page 79 the term gallop rhythm was justified in this connection.

At auscultation the gallop rhythm was, as a rule, perceived as a duplication of the first heart sound.

In all cases, the extra sound was registered after the P wave

in the electrocardiogram. Owing to the tachycardia, it was impossible to decide whether it was a third heart sound or an auricular sound. Therefore, in order to be able to determine the nature of the gallop rhythm, a reduction of the pulse frequency was required. This was achieved by means of carotid sinus pressure in half of the cases. In these cases the gallop rhythm proved to be a third sound gallop (see Fig. 5).

The gallop rhythm was earliest perceived on the 3rd day and latest on the 12th day after the injection, usually on the 4th to 6th day. In 1 case it disappeared already after 2 days but, as a rule, not until after 2—4 weeks, in 5 cases remaining during the whole period of observation. In a few cases, it might have been absent on a certain occasion, only to reappear later. Sometimes, the frequency as well as the amplitude could be seen to diminish successively until the extra sound could finally no longer be registered (see Fig. 9).

The röntgenogram:

A Röntgen examination of the heart was performed in 24 cases. 13 of these disclosed a dilatation which was ascertainable in the frontal as well as in the lateral view and could be described as marked in 7 cases and moderate in 6. The dilatation appeared earliest on the 4th day and latest on the 9th day after the injection, usually occurring on the 4th to 6th day, most often even having reached a maximal development by then. In about half of the cases it remained for the whole of the observation period, but in the other half disappeared more or less completely (see Figs. 7 and 10).

The result of the examination was well in accordance with that of WASSERMEYER and ROHRBACH (1932) who found a röntgenologically ascertainable dilatation on the 5th to 6th day after a coffein-adrenalin injection.

Patho-anatomical examination:

As already mentioned, one of the animals by mistake was not subjected to autopsy, but in all the other cases the heart was examined macro- as well as microscopically. In several cases,

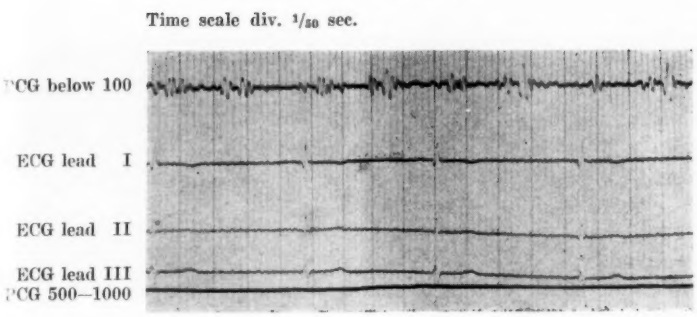


Fig. 5. The same rabbit as in Fig. 6 b. After retardation of the pulse by means of carotid sinus pressure, the gallop sound was found to be a third heart sound.

a more or less distinct dilatation was noticed. The dilatation seemed to have affected chiefly the right ventricle and, next, the left auricle, but also the left ventricle sometimes showed a certain enlargement. Distinct macroscopical changes of the cardiac muscle were observed in several instances, consisting of a diffuse fibrosis or more circumscribed foci principally in the anterior, lateral or posterior wall of the left ventricle, usually closer to the base than to the apex. The macroscopic picture, therefore, rather resembled the one observed by earlier authors (FLEISHER and LOEB 1909, JOHNSON and SIEBERT 1927—28).

The weight of the heart varied between 5.6 and 12.3 grammes. When it was divided by the body weight in Kg, a figure was obtained which formed a minimum of 2.07 and a maximum of 3.88, the mean figure being 3.01. The corresponding values with regard to the 10 control animals were 1.89, 2.77 and 2.44, which were in accordance with data from the literature (CLOETTA 1908). Thus, a certain hypertrophy apparently occurred in the hearts of the animals subjected to treatment as compared with the normal material, in spite of the fact that the latter was too restricted for statistical proof. This is in agreement with observations made by FLEISHER and LOEB (1909) and GRUBER et al. (1933). In contrast, WASSERMEYER and ROHRBACH (1932) were unable to detect any hypertrophy in their cases.

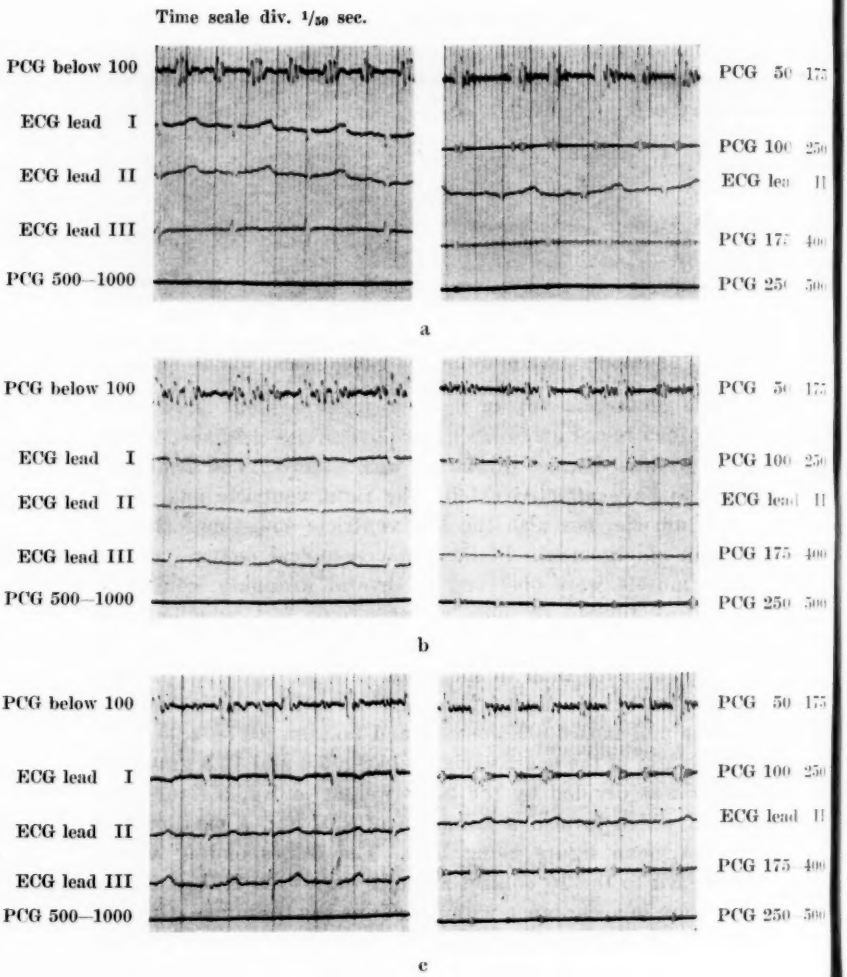
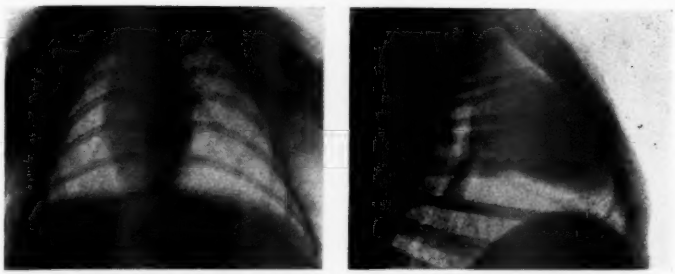
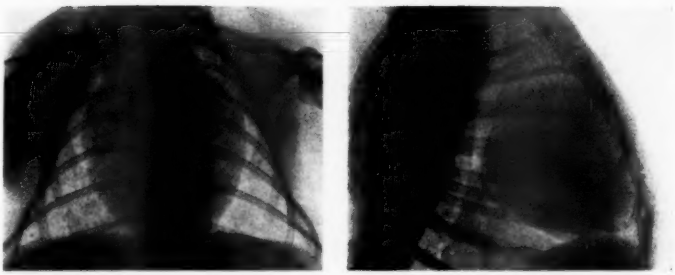


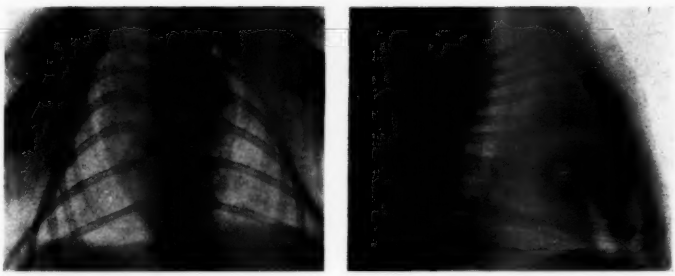
Fig. 6 a—c. Rabbit 28. ECG and PCG (a) before, (b) 5 days after and (c) 7 weeks after coffein-adrenalin injection. In (b) T_1 is inverted and a gallop rhythm is recorded up to the frequency range of 100—250 cycles per second. In (c) the gallop rhythm is lacking while the changes in the ECG remain. Cp. also Fig. 7.



a



b



c

Fig. 7 a—c. The same animal as in Fig. 6. The size of the heart shadow in frontal and lateral view, (a) before, (b) 4 days after and (c) 7 weeks after coffein-adrenalin injection. Marked dilatation on the 4th day, which has decreased considerably 7 weeks later. — The figures are reproduced in $^1/_2$ scale.

Fig. 8 a. A microscopical section from the cardiac muscle of a normal rabbit. Between the muscle fibres, vascular lumina are seen more or less filled with blood corpuscles. The interstitial connective tissue is exceedingly sparse and does not appear in the picture.

The macroscopical examination revealed no stasis in the lungs or in the liver.

For the purpose of microscopical examination, an incision was made straight through the two ventricles, perpendicular to the longitudinal axis of the heart and about 10 mm below the border between auricle and ventricle.

13 of the 29 examined animals disclosed considerable changes, while they were only slight or moderate in 9. These changes were throughout of a similar appearance, consisting, partly, of a diffuse and, partly, of a more focal increase of interstitial connective tissue, mainly in the left ventricle. The connective tissue was in a few cases of a fairly loose consistency but in most cases firmer, in accordance with the comparatively long period of observation, usually lasting for 5—8 weeks (see Fig.

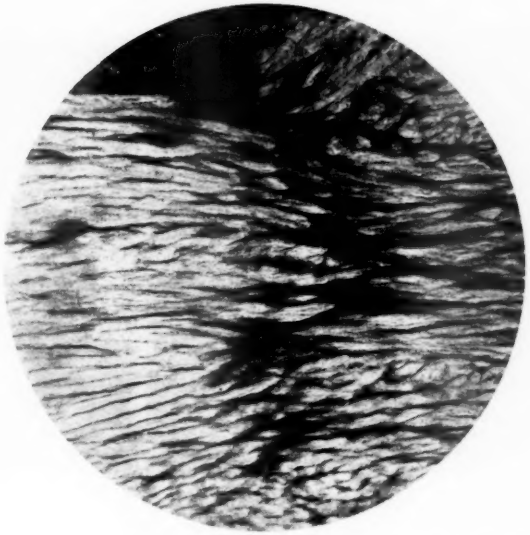


Fig. 8 b. Rabbit 29. Killed 7 weeks after coffein-adrenalin injection. Microscopical section from the cardiac muscle. The same magnification as in Fig. 8 a. The interstitial connective tissue is strongly increased, here and there accumulating in scars. Considerable hypertrophy of the muscular fibres.

8 b). A distinct, often marked hypertrophy of the muscle fibres was present in several cases.

The result of the microscopical examination concurred with what had already been described by earlier investigators (FLEISHER and LOEB 1909, JOHNSON and SIEBERT 1927—28).

Gallop rhythm at experimentally produced myocardial damage in rabbits

The changes in the heart due to the coffein-adrenalin injection may be summarized in the following manner:

Among the 30 animals who survived the injection, the heart in 1 case by mistake was not subjected to a patho-anatomical examination, nor was a Röntgen examination performed.

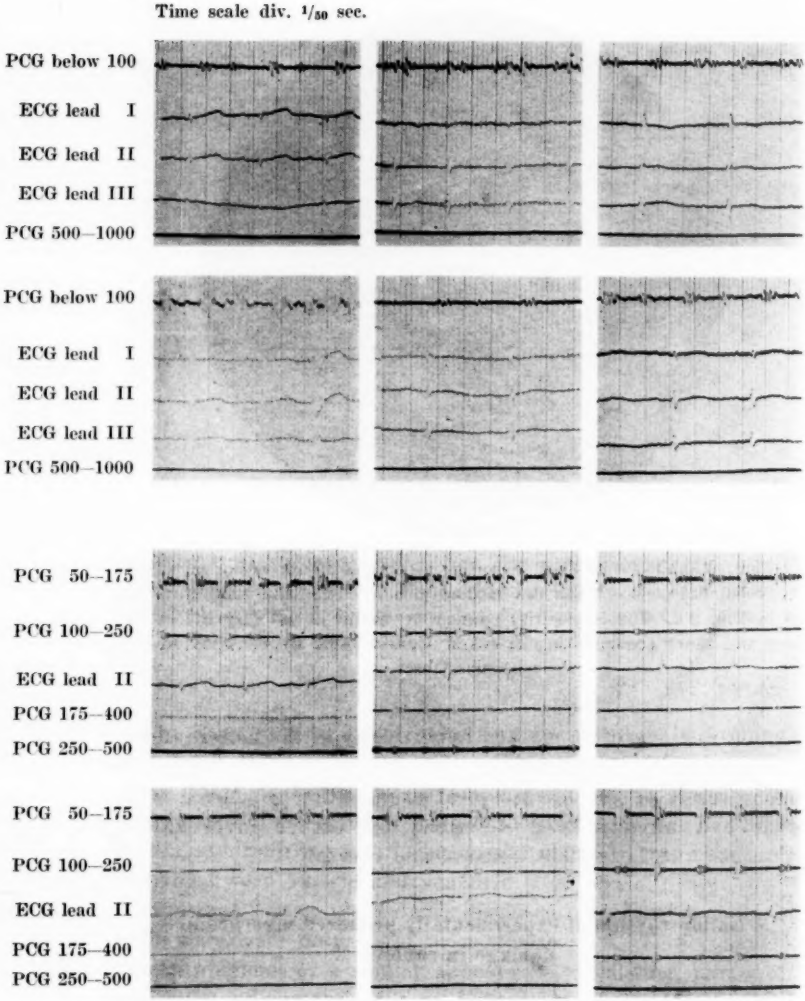


Fig. 9 a—f. Rabbit 29. ECG and PCG (a) before, (b) 4 days after, (c) 11 days after, (d) 3 weeks after, (e) 5 weeks after and (f) 7 weeks after coffein-adrenalin injection. In (b) a distinct gallop rhythm is registered. The extra sound in the two lowest frequency ranges is more pronounced than the second heart sound, and reaches just only up to the frequency range of 250 cycles per second. In (c) the gallop rhythm is much fainter and has disappeared entirely at later examinations (d—f). Changes in the ECG in the form of inverted T_1 appear and disappear parallel with the gallop rhythm. Cp. Fig. 10.

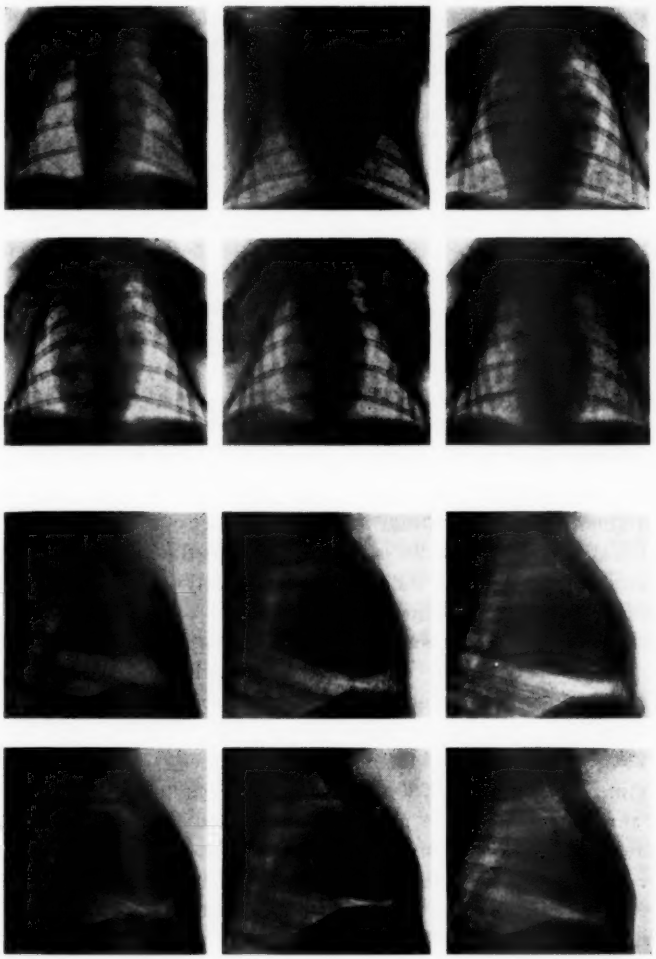


Fig. 10 a—f. The same animal as in Fig. 9. The size of the heart shadow in a frontal and lateral view at the same examinations as in Fig. 9 a—f. Marked dilatation on the 4th day, remaining thus until the 11th day, after which it successively decreases and has completely disappeared after 7 weeks. The dilatation appears and decreases parallel with the gallop rhythm. — The figures 9 and 10 are reproduced in $^2/_3$ scale.

TABLE 8.

Frequency of heart dilatation and gallop rhythm at different degrees of patho-anatomical changes after coffein-adrenalin injection.

Degree of microscopical changes	Total number of cases	Number of cases with	
		dilatation	gallop rhythm
Considerable	10	9	8
Moderate	7	4	2
None	7	0	0

However, the electrocardiogram revealed distinct changes in the S—T segments. A gallop rhythm could not be perceived in this particular case.

In 7 of the 29 remaining cases the microscopical picture showed no pathological signs. The electrocardiogram and the röntgenogram were normal and no gallop rhythm occurred.

Finally, 20 of the 22 animals with distinct microscopical changes in the heart showed pathologically changed electrocardiograms. The Röntgen examination of the heart was performed in 17 cases and disclosed a dilatation in 13. A gallop rhythm was observed in 12 cases and, like the dilatation, principally in the group with the most pronounced patho-anatomical changes (see Table 8).

From the examination it was established that the gallop rhythm, like the dilatation, was a sign of myocardial damage. In the cases where the heart was subjected to röntgenological examination, both these symptoms were found to occur simultaneously to an extent indicating an intimate connection between them. Thus, among 10 cases of gallop rhythm a distinct dilatation was noted in 9. On the other hand, this was not an indispensable condition for the occurrence of gallop rhythm, nor did a gallop rhythm appear in all the cases with dilated hearts.

The relationship between gallop rhythm and cardiac lesions was further displayed by the coincidence of the various signs

after the injection. Both the gallop rhythm, the dilatation and the electrocardiographic changes were most often manifested on the 4th to 6th day (see Figs. 9 and 10 as well as the Case History).

Summary

Myocardial lesions were produced in 23 out of 30 rabbits by means of a method elaborated by Fleisher and Loeb. They were then analysed by means of electrocardiography, phonocardiography, röntgenography and patho-anatomical examinations. An extra sound was ascertained in 16 of the animals. In 12 of these it revealed a higher frequency and a greater amplitude than the extra sounds occasionally registered in the animals not subjected to treatment. Therefore, the term gallop rhythm was considered justified in this connection. In all the cases where the exact nature of the sound was determinable, the extra sound was found to be a third sound gallop.

The heart was submitted to Röntgen examination in 24 instances, a distinct dilatation being observed in 13. The gallop rhythm as well as the dilatation occurred exclusively in the cases with clearly discernible microscopical changes. Furthermore, the two symptoms were manifested simultaneously, to an extent indicating an intimate connection between them.

The electrocardiographic and röntgenologic changes, like the gallop rhythm, as a rule appeared on the 4th to 6th day after the injection and, in several cases, disappeared more or less completely before the animals were killed. The time of observation usually amounted to 5—8 weeks, the patho-anatomical changes, correspondingly, showing different phases of reparation. Only 1 of the animals died spontaneously. The immediate prognosis quoad vitam was, accordingly, favourable.

The examination proved that, by means of the method employed, a distinction was possible between the physiological and the pathological extra sounds and that the latter must be interpreted as signs of myocardial damage.

The gallop rhythm in homo, particularly the third sound gallop, has generally been connected with an abnormal decrease

in myocardial tone, with the result that the ventricular walls are more easily caused to vibrate owing to the sudden inrush of blood from auricle to ventricle. The intimate connection between dilatation and gallop rhythm, as disclosed by the experimental investigation, seems to support this assumption.

CHAPTER IV

The normal phonocardiogram in children

Description of normal cases

The normal cases comprise 150 children aged 3—17 years, half of which were boys. The youngest came from day nurseries and the older ones from elementary schools and municipal secondary schools in Stockholm.

In selecting the normal cases, principal regard was paid to the occurrence of earlier infectious diseases in the anamnesis, particularly those tending to affect the circulatory organs. Accordingly, children who have suffered from diphtheria, scarlet fever or a rheumatic infection have been excluded, as well as those who have had pneumonia or some other more serious infectious disease, protracted angina with a high temperature or repeated pharyngeal infections. On the other hand, children who have earlier been subjected only to mild infections, such as the common cold or an uncomplicated measles, the whooping-cough, the mumps or the chicken-pox, have been included in the normal material.

As to the physical condition of the child at the time of the examination, no regard has been paid to its constitution, i. e. whether it was of strong or slight build, fat or slender, etc. By means of hemoglobin determinations acc. TALLQVIST, it has been checked that no pronounced anemia occurred; nor was albuminuria found in any case. In all but 10 cases, where the children refused, the sedimentation rate was determined acc. WESTERGREN (STRÖM's micro-method being adopted for the smallest children). The sedimentation rate exceeded 15 mm in a few cases which were, consequently, not included in the normal material notwithstanding the fact that the children otherwise

seemed quite healthy. In addition, a small number were excluded whose electrocardiograms could not be accepted as fully normal. Thus, a prolonged conduction time was observed in some of the electrocardiograms (i. e. > 0.18 seconds), others having a relative left axis deviation, and yet others deviations of the S—T segments of more than 1 mm.

Among the remaining 150 normal cases, a faint systolic murmur was perceived over the heart in 27. This was demonstrated by Mc Kee (1938 a) and Mannheimer (1940) as an altogether normal phenomenon. Measurements of the blood pressure have on the whole, shown normal values. Only in a few cases did the systolic blood pressure lie slightly above normal (130—145 mm). However, only one determination was made in each case. The diastolic pressure was normal in all cases (i. e < 80 mm Hg).

Analysis of the normal phonocardiogram in children

In order to ensure as normal a heart activity as possible and to reduce the influence of mental factors, the children have been made to rest for 20 minutes before registration. The registration was performed with the microphone over the apex (ictus) and the base (second intercostal space to the left of the sternum), as well as in the erect position with the microphone at the apex.

A complete analysis of the phonocardiogram includes a determination of the amplitude of the heart sounds and the murmurs, their frequency and duration. However, it is often difficult to determine the duration exactly. This applies especially to the frequency range of < 100 cycles per second where the intervals between the sounds are seldom quite »silent». Since experience has shown that a determination of the duration is of very slight practical significance in judging normal and pathological cases, this has not been performed in the present investigation.

By simultaneously registering the heart sounds and the murmurs in several different frequency ranges, a measure has been obtained of the occurrence of overtones of the various sound phenomena.

The amplitudes are often, even in the same individual and

at the same examination, of varying size in the different cardiac cycles. This applies, above all, to the third sound and the auricular sound. The mean of three successive sounds has therefore been calculated in order to determine the amplitudes. Amplitudes < 1 mm have been regarded as non-existent. Only the three lowest frequency ranges have been included.

The result of the amplitude measurements has been subjected to statistical analysis with regard to the mean and range of variation. The latter has been defined in Tables 10 a and b on the basis of $2\,\sigma$, $2\,^1/_2\,\sigma$ and $3\,\sigma$.

At normal distribution, the probability of a value being normal is comparatively great within $\pm\,2\,\sigma$ but decreases the more $3\,\sigma$ is approached, above which limit it practically equals 0. As is evident from Tables 10 a and b, the distribution of the amplitudes of the heart sounds is however skew. The amplitude of the third sound in the frequency range of < 100 cycles per second may be mentioned as an example of this. The value of $M - \sigma$ here inconsiderably exceeds 0. Still, the amplitudes cannot be less than 0. MANNHEIMER (1940) showed that, in spite of the skew distribution, only a small number of values passed $M + 3\,\sigma$. He explained this as due to the fact that a negative excess occurs simultaneously which, to some extent, neutralizes the effect of the skew distribution. The abnormal distribution should, according to him, conceivably be caused by the lack of homogeneity in the material and, above all, owing to the varying thickness of the thorax. The amplitudes are, for instance, throughout lower in girls, during and after puberty, with their more or less developed mammary glands.

On account of the lack of normality in the distribution, the results of the statistical analysis must be judged with some caution. However, the rules already mentioned, applying to a normal distribution, should be good enough for practical use. In the following, therefore, the range of variation will be denoted in $M \pm 2\,\sigma$ and, in brackets, $M \pm 3\,\sigma$.

As regards the relationship of the heart sounds to the electrocardiogram, only the distance of the third sound from the second sound and the distance of the auricular sound from the P wave have been considered to be of any interest in this investigation.

Finally, a few words may be said regarding the zero values. It may at times be hard to decide whether they should be included in the statistical analysis or not. In the case of an interval between sounds, e. g., between the second and third heart sounds, naturally only the positive ones should be taken into account. When a third sound has not been registrable in a certain case, there is no justification for equalling this interval to zero. A more sensitive apparatus might, perhaps, have disclosed the sound and an interval far above zero would have been obtained. Thus, the statistical calculation will, in this case, be carried out exclusive of the zero values.

With regard to the amplitudes, the situation is different. These may sometimes approach to zero and, in some instances, escape registration with the apparatus employed. The amplitude of the third sound in the frequency range of < 100 cycles per second (Table 9) may be offered as an example of this. In this case it is quite natural to include the zero values in the statistical calculations. However, when the latter are very frequent in the material, this principle can hardly be maintained. For instance, in determining the amplitude of the third sound in the frequency range of 50—175 cycles per second (Table 9), the statistical calculation could well be performed both with the exclusion and inclusion of the zero values. In a given case, it will then have to be decided which of the values are to be used.

The first and second sounds.

Figs. 11 and 12 show the phonocardiograms from normal children. It will be seen from them that the first as well as the second sound consists of a number of more or less irregular vibrations with comparatively large amplitude in addition to overtones of a lower intensity. The difference in amplitude between the two lowest frequency ranges (< 100 and 50—175 cycles per second), on the one hand, and the higher ones, on the other, is in reality greater than illustrated in the figures, the amplification in the frequency range of 100—250 cycles per second being 10 times larger than in the two first-mentioned ranges, that of the still higher ones being 30 times larger. The

TABLE 9.

Measurable amplitudes of heart sounds and murmurs in different frequency ranges.

Normal cases.

n_1 = number of cases including 0 figures.
n_2 = » » » excluding 0 figures.

Frequency range cycles per second	n_1	n_2					
		First sound	Second sound	Third sound	Auricular sound	Systolic murmur	Diastolic murmur
< 100	150	150	150	117	75	.	.
50—175	150	150	150	12	0	74	0
100—250	150	150	150	0	0	70	0
175—400	150	124	148	0	0	8	0
250—500	150	55	121	0	0	0	0

amplitudes in the three lowest frequency ranges are recorded in Table 10 a.

The first and second sounds are registered in all cases up to the frequency range of 100—250 cycles per second, and in a large percentage even higher (Table 9). They invariably consist of overtones, to a lesser or greater extent, which will explain why they are audible under all circumstances, even when the amplitude of the »fundamental tone» is small, as is often the case, above all in girls during puberty, owing to the development of the mammary glands. In contrast, the physiological auricular sound is practically never audible and the third sound but rarely so, in spite of the fact that the amplitude in the frequency range of < 100 cycles per second sometimes approaches that of the first and second sounds. This is due to the almost complete lack of overtones in these sound phenomena (Table 9).

The third sound.

The third sound, in contrast to the two previous sounds, is a sound phenomenon of extremely low frequency, being registered as a rule only in the frequency range of < 100 cycles per second

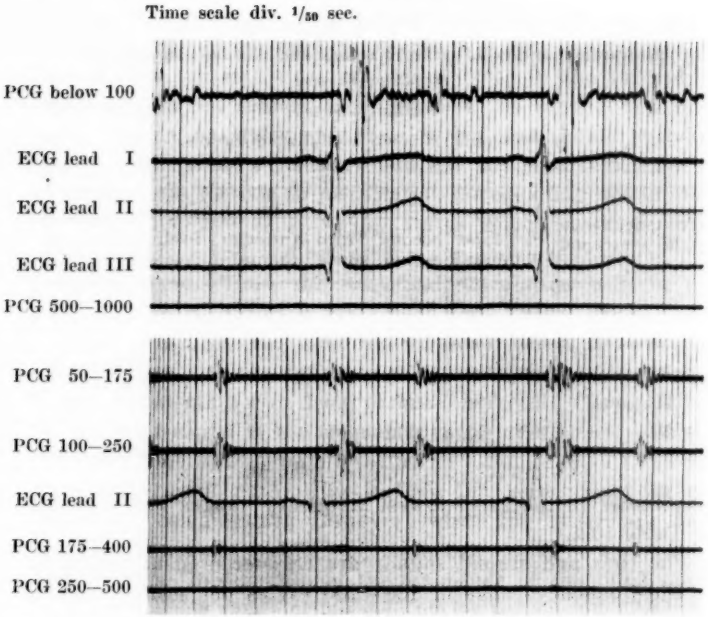


Fig. 11. Normal case no. 71. A boy, aged 5 years. Normal ECG and PCG: Third heart sound only registered in the frequency range of < 100 cycles per second where it has a considerably lower amplitude than the first and second heart sound. A physiological systolic murmur.

(see Figs. 11 and 12). It appears with one or a few vibrations after the second sound and at a distance from the beginning of the latter of on an average 0.143 ± 0.0015 seconds, $\sigma = 0.016$, with a normal variation range of 0.11—0.18 (0.10—0.19) seconds.

By means of the phonocardiographic method employed here, the third sound has been registered in 78.0 ± 3.4 per cent. The mean amplitude equals, inclusive of the zero values, 13.7 ± 1.0 dynes/cm^2, $\sigma = 12.5$, and the normal range of variation is 0—39 (0—51) dynes/cm^2. When the zero values are excluded, the mean will of course be somewhat greater or 17.5 ± 1.1 dynes/cm^2, while the range of variation will, on

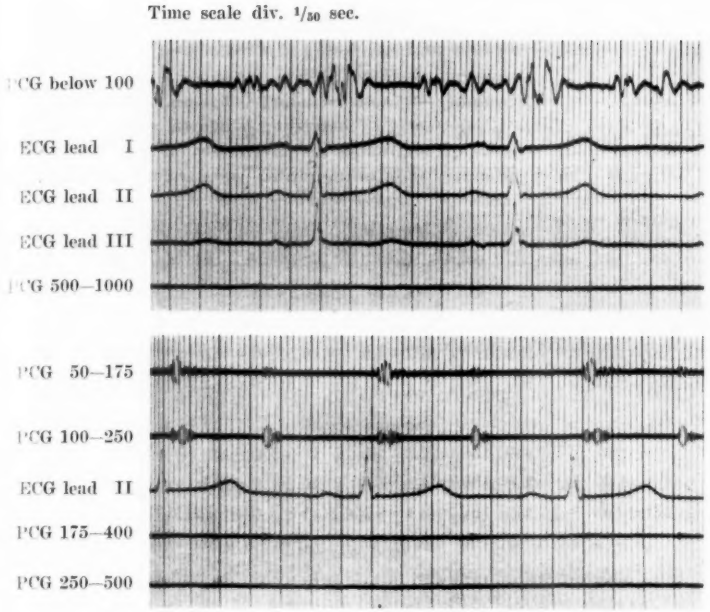


Fig. 12. Normal case no. 40. A girl, aged 14 years. Normal ECG and PCG: The third heart sound has the same amplitude as the second one but is only registered in the frequency range of < 100 cycles per second. Owing to the varying length of diastole, the third sound in certain cycles occurs before, in others after the P wave. A physiological auricular sound is recorded in the frequency range of < 100 cycles per second immediately before the Q wave and passes directly over to the first sound. A physiological systolic murmur.

the other hand, remain practically the same, i. e. 0—41 (0—52) dynes/cm^2 (Table 10).

Only in 12 cases, i. e. 8.0 $\pm$ 2.2 per cent, has the third sound attained the frequency range of 50—175 cycles per second. The amplitude amounted to 4.5 $\pm$ 0.58 dynes/cm^2, $\sigma = 2.0$, when only the positive values were included. The range of variation equalled 0.5—9 (0—11) dynes/cm^2. However, a certain degree of caution is advisable in judging these values owing to the limited number of determinations.

TABLE 10 a.

Amplitudes of heart sounds in dynes/cm².

Normal cases.

n_1 = number of cases including 0 figures.

n_2 = » » » excluding 0 figures.

Statistical characteristics of amplitudes are given only on the basis of n_1.

$M \pm \varepsilon(M)$ = mean ± standard error of the mean. σ = standard deviation.

Frequency range cycles per second	n_1	n_2	$M \pm \varepsilon(M)$	σ	$M \pm 2\sigma$	$M \pm 2.5\sigma$	$M \pm 3\sigma$
First sound							
< 100	150	150	52.9 ± 2.6	32.4	0 – 118	0 – 134	0 – 150
50 – 175	150	150	25.3 ± 1.6	19.5	0 – 64	0 – 74	0 – 84
100 – 250	150	150	1.7 ± 0.11	1.3	0 – 4.3	0 – 5.0	0 – 5.6
Second sound							
< 100	150	150	32.8 ± 1.5	17.9	0 – 69	0 – 78	0 – 87
50 – 175	150	150	24.8 ± 1.3	16.2	0 – 57	0 – 65	0 – 73
100 – 250	150	150	2.4 ± 0.15	1.8	0 – 6.0	0 – 6.9	0 – 7.8
Third sound							
< 100	150	117	13.7 ± 1.0	12.5	0 – 39	0 – 45	0 – 51
50 – 175	150	12	0.36 ± 0.11	1.3	0 – 3.0	0 – 3.6	0 – 4.3
Auricular sound							
< 100	150	75	6.3 ± 0.67	8.3	0 – 23	0 – 27	0 – 31

In no single case was the third sound registrable in the higher frequency ranges, which is in fair conformity with the results obtained by MANNHEIMER (1940). He was able to register a third sound in the frequency range of 100—250 cycles per second in only 2 cases out of 135 normal children.

At auscultation, the third sound has been perceptible more or less distinctly in 12.0 ± 2.7 per cent.

The auricular sound.

The auricular sound, like the third sound, is of a very low frequency and is registered only in the frequency range of < 100 cycles per second (see Fig. 12). It appears with one or a few slow vibrations after the P wave and at a distance from

TABLE 10 b.

Amplitudes of third sound and auricular sound in dynes/cm².

n = number of cases excluding 0 figures.

Frequency range cycles per second	n	$M \pm \varepsilon(M)$	σ	$M \pm 2\sigma$	$M \pm 2.5\sigma$	$M \pm 3\sigma$
			Third sound			
< 100	117	17.5±1.1	11.5	0 −41	0−46	0−52
50−175	12	4.5±0.58	2.0	0.5− 8.5	0− 9.5	0−10.5
			Auricular sound			
< 100	75	12.7±0.86	7.5	0 −28	0−31	0−35

the beginning of this wave of on an average 0.122 ± 0.0013 seconds, $\sigma = 0.011$, the normal range of variation being 0.10—0.14 (0.09—0.16) seconds.

The auricular sound is at times clearly separated from the subsequent first sound by a short interval, but the vibrations may also often pass directly into those of the first sound. It may then be a complicated matter to decide where the auricular sound ends and the first sound begins. Similarly, a determination of the amplitude may, under these circumstances, be difficult. Only vibrations beginning before the onset of the QRS complex have been considered to belong to the auricular sound.

The auricular sound has been registered in half of the normal cases in the frequency range of < 100 cycles per second by means of the apparatus employed here (Table 9).

The mean amplitude equals 6.3 ± 0.67 dynes/cm², $\sigma = 8.3$ and the range of variation is 0—23 (0—31) dynes/cm². When the zero values are excluded, a greater mean is obtained but the range of variation remains practically the same (Table 10).

The auricular sound has not been registered in any single case in the higher frequency ranges.

The murmurs.

Faint systolic murmurs have been registered in approximately half of the cases in the frequency ranges of 50—175 and 100

TABLE 11 a.

Amplitudes of heart sounds in dynes/cm² in boys and girls at different ages. Frequency range < 100 cycles per second.

Normal cases.

n=number of cases including 0 figures. $M \pm \varepsilon(M)$=mean±standard error of the mean. σ=standard deviation.

Age and sex	n	$M \pm \varepsilon(M)$	σ	n	$M \pm \varepsilon(M)$	σ
	First sound			Second sound		
Boys 3– 7 years	25	48.3±5.9	29.3	25	34.2±3.6	18.0
» 8–12 »	25	59.3±8.9	44.3	25	37.2±3.7	18.7
» 13–17 »	25	65.6±5.2	26.2	25	36.7±3.5	17.7
Girls 3– 7 years	25	63.0±6.6	32.9	25	37.2±3.1	15.6
» 8–12 »	25	51.3±5.7	28.7	25	31.0±3.7	18.6
» 13–17 »	25	30.0±3.2	16.1	25	20.4±2.9	14.3
Both sexes 3– 7 years	50	55.7±4.5	31.7	50	35.7±2.4	16.8
» » 8–12 »	50	55.3±5.3	37.2	50	34.1±2.6	18.7
» » 13–17 »	50	47.8±4.0	28.1	50	28.6±2.5	17.9
	Third sound			Auricular sound		
Boys 3– 7 years	25	14.0±2.7	13.3	25	8.4±2.1	10.7
» 8–12 »	25	15.0±2.5	12.6	25	6.6±1.3	6.4
» 13–17 »	25	12.6±2.5	12.6	25	6.4±1.7	8.3
Girls 3– 7 years	25	19.2±2.4	11.8	25	6.6±1.4	7.0
» 8–12 »	25	16.0±2.8	13.8	25	8.4±2.1	10.3
» 13–17 »	25	5.2±1.2	5.8	25	1.6±0.72	3.6
Both sexes 3– 7 years	50	16.6±1.8	12.7	50	7.5±1.3	9.0
» » 8–12 »	50	15.5±1.8	13.1	50	7.5±1.2	8.5
» » 13–17 »	50	8.9±1.5	10.4	50	4.0±1.0	6.8

—250 cycles per second (Table 9). The occurrence of murmurs in the frequency range of < 100 cycles per second is, as a rule, impossible to establish since the intervals between the sounds are rarely sufficiently »silent».

In 8 cases, i. e. 5.3 ± 1.8 per cent, the systolic murmur has been followed up to the frequency range of 175—400 cycles per second. On the other hand, a systolic murmur with a higher frequency has not been registrable in the normal cases.

No diastolic murmurs occur in the normal material.

TABLE 11 b.

Amplitudes of third sound and auricular sound in dynes/cm² in boys and girls at different ages. Frequency range < 100 cycles per second.

n = number of cases excluding 0 figures.

Age and sex	Third sound			Auricular sound		
	n	$M \pm \varepsilon(M)$	σ	n	$M \pm \varepsilon(M)$	σ
Boys 3— 7 years	19	18.4±2.8	12.3	14	15.1±2.7	10.3
» 8—12 »	22	17.1±2.6	12.0	15	11.1±1.1	4.3
» 13—17 »	17	18.5±2.7	11.0	12	13.3±2.1	7.2
Girls 3— 7 years	23	20.8±2.2	10.7	14	11.8±1.3	4.9
» 8—12 »	20	20.0±2.8	12.5	15	14.0±2.5	9.8
» 13—17 »	16	8.1±1.3	5.3	5	7.8	—
Both sexes 3— 7 years	42	19.7±1.8	11.4	28	13.4±1.5	8.1
» » 8—12 »	42	18.5±1.9	12.2	30	12.5±1.4	7.6
» » 13—17 »	33	13.5±1.8	10.1	17	11.6±1.6	6.8

The amplitudes of the heart sounds in boys and girls in different age-groups.

In order to examine whether there is any difference in amplitude between older and younger children or between boys and girls during puberty, the normal material has been divided into three groups comprising the ages 3—7, 8—12 and 13—17 years. This examination dealt only with the frequency range of < 100 cycles per second, the result of which is shown in Table 11.

The 13—17-years-olds throughout showed lower amplitudes than the other age-groups. The difference is statistically significant with regard to the third sound, but only when the zero values are included. When they are excluded, the difference is not even probable owing to the fact that the third sound is registered to a somewhat lesser extent in the older children than in the younger ones.

Furthermore, the table indicates that the difference in amplitude between the 13—17-years-olds, on the one hand, and the 3—7 and 8—12-years-olds, on the other, refers only to the girls in the first-mentioned group. They disclose lower

TABLE 12 a.

Amplitudes of heart sounds in dynes/cm² with microphone at apex and base of heart.

Normal cases.

n_1 = number of cases including 0 figures.
n_2 = number of cases excluding 0 figures.
Statistical characteristics of amplitudes are given only on the basis of n_1.
$M \pm \varepsilon(M)$ = mean ± standard error of the mean. $D \pm \varepsilon(D)$ = difference ± standard error of the difference.

Frequency range cycles per second	Microphone	n_1	n_2	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$	n_2	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$
			First sound			Second sound		
< 100	apex	150	150	52.9±2.6	16.7±3.1	150	32.8±1.5	−0.6±2.1
	base	142	142	36.2±1.7		142	33.4±1.4	
50—175	apex	150	150	25.3±1.6	11.0±1.8	150	24.8±1.3	0.9±1.7
	base	142	142	14.3±0.84		142	23.9±1.1	
100—250	apex	150	150	1.7±0.11	0.7±0.13	150	2.4±0.15	−0.5±0.23
	base	142	138	1.0±0.06		142	2.9±0.17	
			Third sound			Auricular sound		
< 100	apex	150	117	13.7±1.0	10.7±1.09	75	6.3±0.67	4.2±0.77
	base	142	45	3.0±0.43		30	2.1±0.37	

amplitudes than all the other groups and the difference is statistically significant in most of the cases. As regards the first sound, the difference in amplitude between boys in the age-group of 3—7 years and girls in that of 13—17 years is, however, only probable and, as far as the second sound is concerned, it is not even probable in comparing the girls in the age-groups of 8—12 years and 13—17 years. The difference in amplitude of the third heart sound and the auricular sound, as between boys and girls in the age-group of 13—17 years, is probable when the zero values are included. When they are excluded, the difference becomes statistically significant as regards the third sound.

The number of determinations in the various groups is too restricted to permit definite conclusions. However, the result of this examination seems to support the assumption that an increased thickness of the thoracic wall has a modifying effect

TABLE 12 b.

Amplitudes of third sound and auricular sound in dynes/cm² with microphone at apex and base of heart.

n = number of cases excluding 0 figures.

Frequency range cycles per second	Microphone	Third sound			Auricular sound		
		n	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$	n	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$
< 100	apex	117	17.5 ± 1.1	8.1 ± 1.30	75	12.7 ± 0.86	$2.6-1.06$
	base	45	9.4 ± 0.70		30	10.1 ± 0.62	
50—175	apex	12	4.5 ± 0.58		0	0	
	base	0	0		0	0	

on the intensity of the heart sounds in at least the frequency range of < 100 cycles per second, which is in good agreement with MANNHEIMER's findings (1940).

Apart from the difference due to the development of mammary tissue in girls in the age-group of 13—17 years, no definite difference in amplitude has been ascertainable in this examination as between younger and older children. The third sound and the auricular sound do not differ in this respect from the two other sounds, at any rate when judging this normal material.

The amplitudes of the heart sounds with the microphone either at the apex or the base.

It has been propounded earlier that especially the third sound is often heard and registered distinctly only over a limited area over the ictus and more rarely over the base of the heart. For this reason, the normal cases have been examined in the supine position with the microphone applied to the base as well as to the apex.

The results of the investigation are shown in Table 12. From this table it will be seen that the third sound as well as the auricular sound have been registered much less frequently over the base than over the apex. The amplitudes both of these sounds and the first sound throughout disclosed lower values over the base with a statistically significant difference, while

TABLE 13 a.

Amplitudes of heart sounds in supine and erect position in dynes/cm² with microphone at apex.

Normal cases.

n_1 = number of cases including 0 figures.
n_2 = number of cases excluding 0 figures.
Statistical characteristics of amplitudes are given only on the basis of n
$M \pm \varepsilon(M)$ = mean ± standard error of the mean. $D \pm \varepsilon(D)$ = difference ± standard error of the difference.

Frequency range cycles per second	Patient's position	n_1	n_2	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$	n_2	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D$
				First sound			Second sound	
< 100	supine	150	150	52.9 ± 2.6	−6.3 ± 4.1	150	32.8 ± 1.5	−3.7 ±
	erect	148	148	59.2 ± 3.2		148	36.5 ± 1.8	
50–175	supine	150	150	25.3 ± 1.6	−8.9 ± 2.4	150	24.8 ± 1.3	−2.5 ±
	erect	149	149	34.2 ± 1.8		149	27.3 ± 1.4	
100–250	supine	150	150	1.7 ± 0.11	−0.5 ± 0.17	150	2.4 ± 0.15	−0.4 ±
	erect	149	149	2.2 ± 0.13		149	2.8 ± 0.15	
				Third sound			Auricular sound	
< 100	supine	150	117	13.7 ± 1.0	10.5 ± 1.27	75	6.3 ± 0.67	6.06 ±
	erect	148	24	3.2 ± 0.79		3	0.24 ± 0.15	

no difference could be observed as regards the second sound. When the zero values were excluded, the difference was still significant as regards the third sound, but not even probable in the case of the auricular sound. The result therefore confirms the earlier conception that the third sound is best audible at the apex.

The amplitudes of the heart sounds in the erect and in the supine position with the microphone applied to the apex.

According to data in the literature in this field, the third sound should be best audible, or exclusively so, in the supine position. In fact, the experience gained during the work at the heart station has also shown that the third sound as well as the auricular sound generally has a larger amplitude in the supine than in the erect position. The examination of the normal

TABLE 13 b.

Amplitudes of third sound and auricular sound in dynes/cm² in supine and erect position with microphone at apex.

n = number of cases excluding 0 figures.

Frequency range cycles per second	Patient's position	Third sound			Auricular sound		
		n	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$	n	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$
< 100	supine	117	17.5±1.1	−2.5±3.4	75	12.7±0.86	
	erect	24	20.0±3.2		3	12.0	
50—175	supine	12	4.5±0.58		0	0	
	erect	1	6.0		0	0	

cases has been performed also with particular regard to this circumstance. The results are evident from Table 13.

Table 13 shows how the third sound as well as the auricular sound have been registered far less frequently in the erect than in the supine position. The first and second sounds throughout reveal somewhat larger amplitudes in the erect position. However, the difference is statistically significant or probable only as regards the first sound in the frequency ranges of 50—175 and 100—250 cycles per second. On the other hand, the amplitudes of the third sound and the auricular sound are considerably lower in the erect than in the supine position, that is to say, only when the zero values are included. When these are excluded, no difference in amplitude is obtained. The very cases, where the third sound and the auricular sound have been registered also in the erect position, have referred almost exclusively to children in the age-group of 3—7 years.

Thus the examination has confirmed the conception that the third sound is only, or best, audible in the supine position.

Summary

The normal phonocardiogram in children has been studied with particular reference to the amplitudes of the heart sounds in the frequency ranges of < 100, 50—175 and 100—250 cycles per second. They have then been subjected to statistical

analysis with regard to the mean and the range of variation. The investigation shows that the third sound and the auricular sound are sound phenomena of very low frequency, not being registered in any single case within the frequency range of 100—250 cycles per second, while the first and second sounds invariably consist of overtones to a greater or smaller extent. The auricular sound occurs exclusively in the frequency range of < 100 cycles per second while the third sound in less than 10 per cent of the cases is recorded also in the frequency range of 50—175 cycles per second.

A difference in the size of the amplitude as between older and younger children is only observed insomuch as girls, during and after puberty, throughout reveal fainter sound phenomena than the other groups which is explained as due to their thicker thoracic wall.

The first sound as well as the third one and the auricular sound disclose greater amplitudes over the apex than over the base. The difference is, apparently, most marked in the case of the third sound. This is in accordance with data in the literature stating that the third sound is best audible at the apex.

The conception that the third sound is best heard in the supine position is confirmed moreover by a comparison between the amplitudes in the erect and in the supine position. The amplitude of the third sound and the auricular sound is considerably less when standing up than when lying down.

CHAPTER V

Definition of gallop rhythm in children by means of calibrated phonocardiography

As mentioned earlier, the gallop rhythm has been found to occur owing to an accentuation of the physiological third sound and the auricular sound, respectively. However, it has not been possible to distinguish between the physiological and pathological extra sounds by means of the phonocardiographic methods hitherto employed. On the other hand, such a distinction can be made with the aid of calibrated phonocardiography.

In the preceding chapter, the normal phonocardiogram in children was studied. On the basis of these observations, the *gallop rhythm in children* is defined in the following way:

Third sound gallop is present —

when the third sound is registered in the frequency range of 100—250 cycles per second or higher, or

when the amplitude of the third sound in the frequency range of $<$ *100 cycles per second exceeds 50—60 dynes/cm*^{2} *and in the frequency range of 50—175 cycles per second 10—12 dynes/cm*^{2}.

Auricular sound gallop is present —

when the auricular sound is registered in the frequency range of 50—175 cycles per second or higher, or

when the amplitude of the auricular sound in the frequency range $<$ *100 cycles per second exceeds 30—40 dynes/cm*^{2}.

No general applicability or exactness can be claimed of course for the definition of gallop rhythm offered here. Though the method is calibrated, certain technical difficulties must be taken into account as in all complicated apparatus. Furthermore, the

frequency ranges have been selected arbitrarily. Another selection would give different values of the amplitudes. Finally, the normal material is fairly small. No definite conclusions may therefore be drawn with regard to a distinction between the normal and the pathological.

In spite of these reservations, calibrated phonocardiography has decided value as an objective method for distinguishing between physiological and pathological extra sounds, as will be shown in the following.

CHAPTER VI

Gallop rhythm in childhood. Analysis of 104 cases

Introduction

During the years 1939 to 1945, about 5000 children were examined at the heart station of the Crown Princess Lovisa's Children's Hospital by means of calibrated phonocardiography. These children were treated either clinically or policlinically at the hospital. The reason for examination was naturally a more or less well founded suspicion of cardiac disease. The only sign was frequently a systolic murmur which could not be satisfactorily analysed merely by auscultation. However, faint systolic murmurs are to a great extent a normal occurrence and the material has, therefore, come to include also a large number of children with healthy hearts.

A gallop rhythm was diagnosed in 104 cases during the above-mentioned period. In 71 of these cases the extra sound was registered before the P wave in the electrocardiogram (see Fig. 13, page 89), the distance from the beginning of the second sound being, in all cases but one, within the normal range of variation of the distance of the third sound from the second (see page 68). In 3 cases, the extra sound simultaneously occurred at the upper limit of the normal distance between the second sound and the opening snap (cp. page 33). Since, however, nc signs of mitral stenosis were noted in these cases, and considering that the extra sound was typical of a third sound, the possibility of an opening snap could be excluded. In all these cases, the gallop rhythm was, accordingly, interpreted as a third sound gallop.

In the remaining 33 cases, the extra sound was registered after the P wave in the electrocardiogram. 7 of these cases

disclosed a distance from the second sound which lay within the normal range of variation of the distance of the third sound from the second, while the interval from the beginning of the P wave fell considerably below the lower limit of the normal distance of the auricular sound from this wave (see page 71). Since an opening snap was out of the question also in these cases, the gallop sound was probably a third sound which had come to lie after the P wave in the electrocardiogram owing to tachycardia. In 2 cases, on the other hand, the extra sound occurred within the normal range of variation of the interval P-auricular sound, while the distance from the second heart sound exceeded the upper limit of the normal distance of the third sound from this sound. The gallop sound was, therefore, in these 2 cases probably an auricular sound (cp. Fig. 14, page 89). Finally, the extra sound in 24 cases had such a position after the P wave as, possibly, to be due to auricular systole as well as to the rapid ventricular filling. It could, consequently, not be classified more precisely (see Fig. 15, page 90). However, it has occasionally been possible to change a previously undeterminable gallop rhythm into a typical third sound gallop by means of a retardation of the heart frequency by carotid sinus pressure or by the injection of neostigmin. It is therefore quite likely that the extra sound has, in at least a few of these cases, been a third sound. Still, it is also conceivable that it may, in some instances, have been an auricular sound. The extra sound has in certain cases had an appearance (irregular vibrations, long duration) indicating a possible summation of a third sound gallop and an auricular sound gallop occurring simultaneously. However, the term summation gallop has been employed in all cases where the extra sound has offered criteria both of a third sound gallop and of an auricular sound gallop, and where a retardation of the pulse has failed, thus making it impossible to determine the nature of the gallop rhythm. In this connection, the terminology generally adopted has been used.

Thus, judging from the above-mentioned cases, the third sound gallop would be by far the most common form of gallop rhythm in children. In 67 out of 78 cases with a third sound gallop, the third sound was registered in the frequency range of

100—250 cycles per second or higher. However, the amplitudes in the two lowest frequency ranges then often lay within the normal range of variation. On the other hand, in the remaining 11 cases the amplitude of < 100 cycles per second exceeded $M + 3\sigma$ without a definite pathological increase in frequency. Accordingly, there has not invariably been an absolute parallelism between amplitude and frequency of the third heart sound. However, on the whole a sound of fairly high frequency may be said to have a bigger amplitude than a sound with a comparatively low frequency.

An examination of 60 cases of third sound gallop with the third sound registered in the frequency range of 100—250 cycles per second has shown that the auricular sound was only registered, as in the normal cases, in the frequency range of < 100 cycles per second, with on an average the same amplitude as in these. This would signify that the mechanism causing an accentuation of the third sound did not produce a corresponding increase in the amplitude and frequency of the auricular sound in these cases.

The gallop rhythm is perceived almost exclusively in the supine position (see Fig. 13). However, it may at times be heard distinctly even in the erect position, above all in small children. In Table 14, a comparison is made between the amplitudes in the supine and in the erect position in 39 cases of third sound gallop where the third sound has been registered in the frequency range of 100—250 cycles per second. It is evident from the table that the third sound was registered far less often in the erect than in the supine position, above all, in the frequency ranges of 50—175 and 100—250 cycles per second, and that the amplitudes were much smaller in the former position. The difference is statistically significant even when the zero values are excluded.

The fact that the third sound gallop as well as the physiological third sound are weakened or disappear in the erect position cannot be explained by deteriorated conduction of the sounds to the thoracic surface. In such a case, all the sound phenomena should become fainter. The explanation must, in all probability, be sought for rather in the altered physiological conditions

TABLE 14.

Amplitudes of heart sounds in dynes/cm² in supine and erect position with microphone at apex.

39 cases of third sound gallop.

n = number of cases excluding 0 figures. $M \pm \varepsilon(M)$ = mean ± standard error of the mean. $D \pm \varepsilon(D)$ = difference ± standard error of the difference.

Frequency range cycles per second	Patient's position	n	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$	n	$M \pm \varepsilon(M)$	$D \pm \varepsilon(D)$
		First sound			Second sound		
< 100	supine	39	58.2±5.5	0.4±8.5	39	34.3±2.4	−0.4±4.7
	erect	39	57.8±6.5		39	34.7±4.0	
50−175	supine	39	32.2±4.1	−5.6±5.7	39	25.6±2.3	−1.5±3.9
	erect	39	37.8±3.9		39	27.1±3.1	
100−250	supine	39	2.0±0.29	−0.4±0.43	39	2.1±0.22	0.1±0.27
	erect	39	2.4±0.32		39	2.0±0.15	
		Third sound			Auricular sound		
< 100	supine	39	46.2±4.2	26.5±5.1	27	14.3±1.8	
	erect	27	19.7±2.9		1	12.0	
50−175	supine	39	18.5±2.2		0	0	
	erect	9	6.7		0	0	
100−250	supine	39	0.64±0.061		0	0	
	erect	2	0.45		0	0	

occurring in the erect as compared to the supine position. When lying down, the blood flow to the heart is stronger and the diastolic tone of the ventricular walls less. NYLIN (1934), among others, proved the heart volume to be greater in the supine than in the erect position. As pointed out previously, the third sound gallop, like the physiological third sound, is generally considered to be due to a sudden distension of the ventricular wall owing to the rapid inrush of blood at the beginning of diastole. The stronger the flow of blood and the more decreased the tone of the cardiac muscle, the more probable the appearance of the third sound. Thus, the conditions favouring the appearance of this sound are more likely to occur in the supine position.

As a rule, the gallop rhythm is only heard in a limited area over the ictus and, more rarely, over the entire precordium. In

conformity with this, the largest amplitude of the gallop sound has generally been registered over the apex. Even a relatively small deviation of the microphone towards the base has often caused the amplitude to decrease considerably. It is therefore necessary to place the stethoscope sufficiently far out at the apex in order to avoid the risk of failing to notice the gallop rhythm at auscultation.

The gallop rhythm has under ideal conditions been distinctly perceptible in practically all the examined cases, i. e. with the patient lying down and the stethoscope applied over the ictus. The exceptions chiefly concern children with a pronounced tachycardia and loud murmurs which have covered the extra sound. However, by auscultation alone it was often not possible to say whether in a given case a gallop rhythm or a physiological third sound was present. The latter may also, though more exceptionally, be distinctly audible (cp. page 70).

As already stated, the gallop rhythm has above all been studied in elderly persons. It has been found particularly often in the hypertensive-arteriosclerotic diseases, as well as in aortic defects, generally being regarded as a sign of a failing left ventricle. In these cases, the gallop rhythm was supposed to be due to a reduction in tone of the structurally altered ventricular wall. Only a few clinical examinations have been made on children. Even though a gallop rhythm has generally been interpreted also in children as a sign of myocarditis or myocardial damage, such a relationship has not as yet been convincingly demonstrated owing to the impossibility hitherto of distinguishing between physiological and pathological extra sounds at this age.

The cases of gallop rhythm examined here belong to two groups, the first comprising children suffering from certain well-defined diseases, principally heart diseases. The children belonging to the second group have not suffered from such a well-defined disease, but in these cases certain cardiac changes have, nevertheless, often been found by means of special examination methods. The former group comprises 39 and the latter group 65 children. The exact distribution of the material with regard to diagnosis is illustrated in Table 15 below.

TABLE 15.

The distribution of the gallop rhythm cases with regard to diagnosis.

Diagnosis	Number of cases
Rheumatic infection	24
Other infections	6
Congenital heart defects	5
Anemia	4
No well-defined disease	65

Gallop rhythm in rheumatic infection

In 24 cases gallop rhythm has been observed in connection with a rheumatic infection. At the time of the first ascertainment of the gallop rhythm, 17 cases disclosed definite signs of carditis; as far as is known, they had not suffered from any heart disease earlier. A valvular defect was noted in 6 cases. 3 of these had probably had a relapse of the carditis at the time of the examination while the remaining 3 showed no definite manifestations of an active infection. Finally, no definite signs were found in 1 case, whether of a carditis or of a malformation.

Gallop rhythm in rheumatic carditis without a developed valvular defect.

The frequency of gallop rhythm has been studied in all cases of acute rheumatic infection treated at the Crown Princess Lovisa's Children's Hospital during the years 1939—1945. The material comprises 86 children aged 3—14 years. Only definite cases of acute polyarthritis and chorea minor have been included. Since the main purpose of the examination was to study the connection between gallop rhythm and myocarditis, cases were excluded in which it was impossible to determine whether certain signs from the heart, above all murmurs, were manifestations of an active carditis or derived from an already developed defect. However, only 6 cases have been omitted at the examination for this reason.

The great majority of the children were treated for a first

attack of the disease. Only 3 have previously been subjected to an attack without at the time revealing any signs of a cardiac affection. In another 3 cases, choreatic symptoms appeared shortly after their discharge. They were therefore readmitted to the hospital. Owing to the fact that the chorea in these cases appeared in very close connection with the first onset of the disease, it has been interpreted as a manifestation of the same infection and has, accordingly, not been regarded as a new case. In 2 cases, on the other hand, a second attack of the disease followed the first after a considerable period of health; here the second attack has been regarded as a new case. This way, the material has come to comprise 88 cases, 65 of which concerned polyarthritis, 17 chorea and 6 polyarthritis + chorea. A combined electro- and phonocardiographic registration has been performed in all the cases. A Röntgen examination has been carried out in 80 cases. 4 of the children died from the infection, and an autopsy was made in 2 of these cases.

The following heart changes were ascertained:

Electrocardiogram: A prolonged conduction time occurred in 33 cases. 17 of them showed a P—Q interval exceeding 0.20 seconds, and 10 an interval of 0.19—0.20 seconds. In 1 case, the P—Q time remained constantly at 0.20 seconds at repeated examinations. All the rest varied during the time of observation by a minimum of 0.03 seconds.

An elevation or depression of the S—T segment by more than 1 mm in one or several leads occurred in 8 cases, pathologically decreased QRS complexes in 2 cases. Pathological changes in the electrocardiogram have been ascertained in a total of 40 cases, or approximately half the material.

Phonocardiogram: Systolic murmurs were registered to a greater extent than in normal children; in 20 cases they were recorded in the frequency range of 250—500 cycles per second, this being definitely pathological (cp. Tables 9 and 16). In 14 cases, a distinct diastolic murmur was registered. In 2 of these cases, the murmur escaped observation at auscultation. In another 2, on the other hand, a diastolic murmur was heard but could not be registered. In all, 23 cases of pathological murmurs were found.

TABLE 16.

Measurable amplitudes of heart sounds and murmurs in different frequency ranges.

88 cases of acute rheumatic infection in children.

n_1 = number of cases including 0 figures.
n_2 = number of cases excluding 0 figures.

Frequency range cycles per second	n_1	n_2						
		First sound	Second sound	Third sound	Auricular sound	Summation sound	Systolic murmur	Diastolic murmur
< 100	88	88	88	57	38	6	.	.
50–175	88	88	88	20	0	6	71	14
100–250	88	87	88	9	0	6	69	14
175–400	88	63	81	0	0	4	36	14
250–500	88	45	78	0	0	1	20	11

The third sound and the auricular sound were registered to approximately the same extent as in normal children. However, the former attained the frequency range of 100—250 cycles per second in 9 cases, and in another case the third sound was registered with a pathologically increased amplitude in the frequency range < 100 cycles per second. Thus, in these 10 cases the term third sound gallop was justified (Fig. 13). In 1 case, the amplitude of the auricular sound in the frequency range of < 100 cycles per second exceeded the upper limit of the normal range of variation. Therefore, this case represented definition adopted on page 82, was called a summation sound was registered in 6 cases in the frequency range of 100—250 cycles per second or higher which, in accordance with the definition adopted on page 82, was called a summation sound (Fig. 15). Thus, among the 88 cases of acute rheumatic infection a gallop rhythm was registered in a total of 17 cases, or 19 ± 4.2 per cent.

Röntgen: A Röntgen examination of the heart has, as already mentioned, been performed in 80 cases. In 22 of these, the heart disclosed a marked enlargement which, in certain instances, was considerable. Thus, a picture of pericarditis was observed in

On page 88 the eleventh line from the bottom should be exchanged for the following:

an auricular sound gallop (Fig. 14). Finally, an extra sound

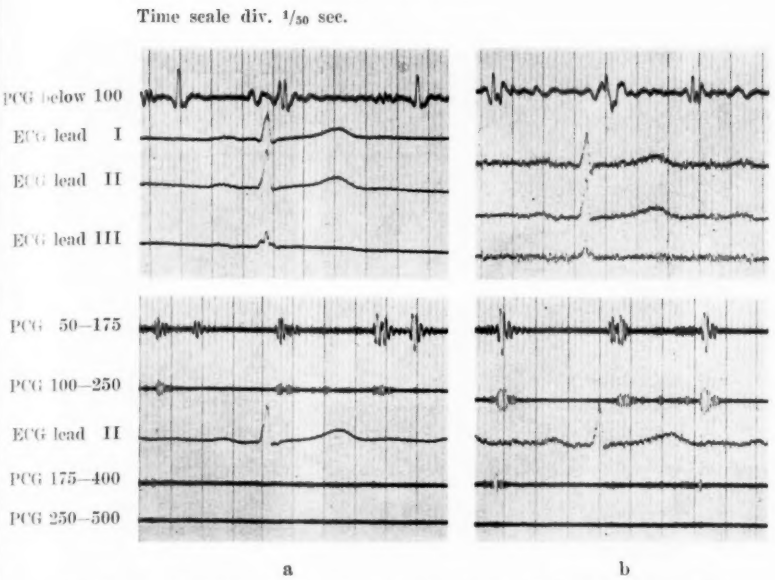


Fig. 13 a and b. ECG 475/43. A boy, aged 8 years. Polyarthritis acuta + endocarditis acuta. ECG and PCG (a) in a supine, (b) in an erect position with the microphone applied to the apex. Third sound gallop with the third sound recorded up to a frequency range of 100—250 cycles per second. A systolic murmur of a high frequency of 50—500 cycles per second which is pathological. The P—Q time was earlier prolonged but is now normal. The gallop rhythm is apparent only in a supine position.

Fig. 14. ECG 216/40. A boy, aged 9 years. Polyarthritis acuta recidivans + endocarditis acuta. ECG and PCG: No definite changes in the ECG. An auricular sound gallop with a pathological amplitude of the auricular sound in the frequency range of < 100 cycles per second. A physiological third heart sound.

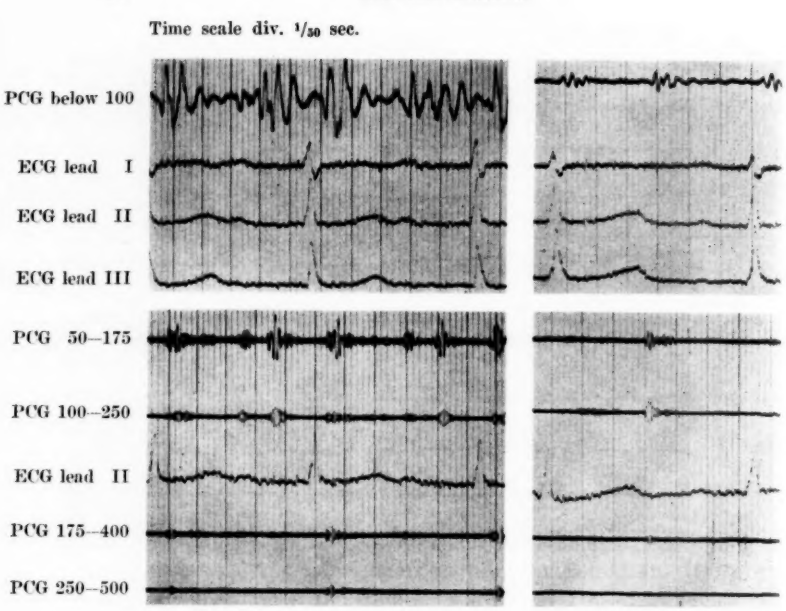


Fig. 15. ECG 971/43. A boy, aged 10 years. Polyarthritis acuta + endocarditis acuta. (a) ECG and PCG at admission: Prolonged P—Q time of 0.24 seconds. Summation gallop with larger amplitude of the extra sound than of the first and second sounds in the frequency ranges of 50—175 and 100—250 cycles per second. Systolic murmur of high frequency of 50—500 cycles per second. (b) ECG and PCG 4 weeks later: P—Q time shorter, equalling 0.18 seconds. The murmur is fainter and the gallop rhythm has disappeared. Cp. Diagram 1.

3 cases, all with stasis in the pulmonary fields. A general dilatation of the heart was observed in 7 cases, 1 of which had stasis. I 4 cases, chiefly the left auricle and ventricle were enlarged, stasis being noticed in 2. Finally, in 8 cases only the left ventricle was enlarged. Apart from these 22 cases showing a marked enlargement, a less conspicuous one was observed in another 17, referring in these cases almost exclusively to the left ventricle. Thus, among the 80 cases subjected to Röntgen examination, a more or less pronounced enlargement was

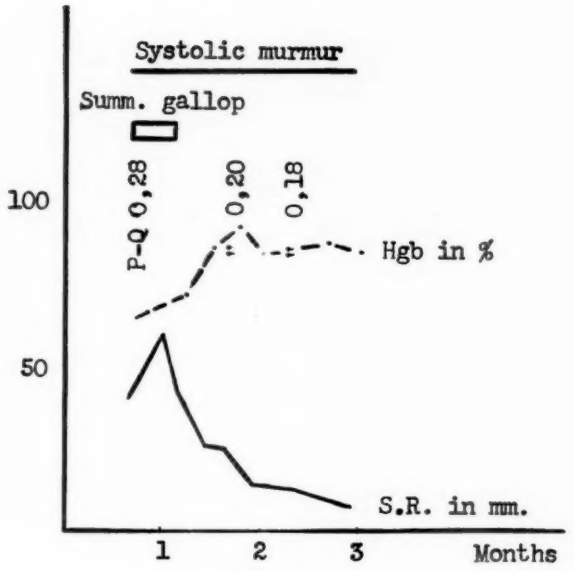


Diagram 1.

ascertained in 39. Only in 18 of these cases could the connection between the enlargement and the infection be proved. As regards the rest of the cases, either only one examination has been performed, or the heart, at repeated examinations, has not disclosed any change of size or configuration. It has therefore been impossible to judge whether the enlargement was the result of the disease under treatment or had occurred earlier. However, it seems most probable that the enlargement was due to the rheumatic infection also in these cases.

2 out of 4 cases who died from the infection were subjected to autopsy. Both disclosed a considerable enlargement of the heart with stasis. In one of these cases a pancarditis occurred, and in the other peri- and myocarditis.

By means of the above-mentioned examination methods, signs of cardiac damage have been ascertained in a total of 58 cases, or approximately $^2/_3$ of the material. This is in fair agreement with the information generally given in the literature regarding

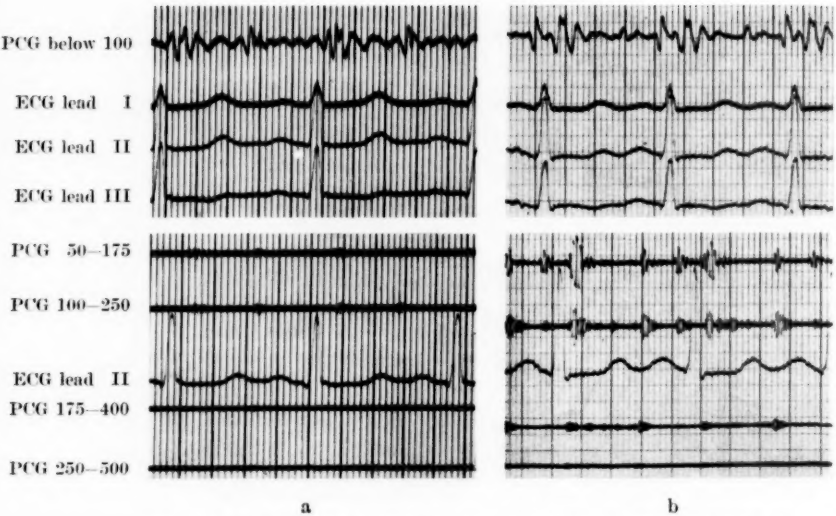


Fig. 16 a and b. ECG 892/44. A girl, aged 13 years. Polyarthritis acuta + myocarditis acuta. (a) ECG and PCG 1 week after admission: But slight changes in the S—T segment. A physiological systolic murmur of 50—250 cycles per second. (b) ECG and PCG 3 months later: Distinctly depressed S—T segments in Lead II. The systolic murmur stronger and of a higher frequency. Gallop rhythm with the extra sound registered up to the frequency range of 100—250 cycles per second. (After a retardation of the pulse by means of an injection of neostigmin, the extra sound was found to be a third heart sound.) Cp Diagram 2.

the frequency of cardic affections in rheumatic fever. In 16 cases changes occurred only in the electrocardiogram, and in 14 of them exclusively in the form of a transient prolongation of the P—Q. In 8 cases a slight enlargement of the left ventricle was the only pathological finding. All transitions are met with from these mild cases to the most severe cases with signs of pancarditis and decompensation.

In order to elucidate the significance of the gallop rhythm in the cases examined here, the frequency of this symptom at different degrees of cardiac involvement has been determined.

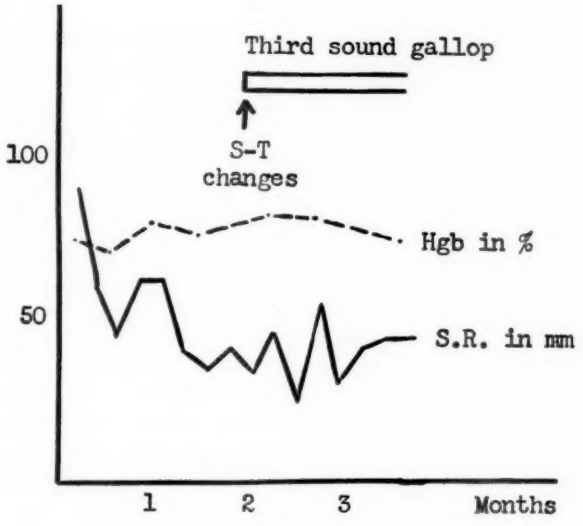


Diagram 2.

For this purpose the material has been divided into three different groups. The first group consists of cases which have revealed distinct signs of endo- or pancarditis, some of which had been decompensated even at rest. In this connection, only definitely pathological murmurs have been considered as signs of endocarditis. In addition, signs of myocarditis occurred in almost every one of these cases. The second group comprises cases displaying electrocardiographic and röntgenological signs of myocardial damage but lacking symptoms of a simultaneous affection of the endocardium or the pericardium. The third group includes all cases where definite signs of cardiac complications could not be elicited. The result of the examination is shown in Table 17.

From Table 17 it will be seen that the gallop rhythm occurred exclusively in cases with signs of cardiac involvement. The experimental examination showed that gallop rhythm appeared in rabbits in connection with myocardial damage. Thus, experimental and clinical experience both favour the assump-

TABLE 17.

Frequency of gallop rhythm at different degrees of cardiac involvement in acute rheumatic infection in children.

Degree of cardiac involvement	Total number of cases	Number of cases with gallop rhythm
Endo (-myo) carditis or pancarditis	25	16
Myocarditis	33	1
No definite signs of carditis	30	0

tion that the gallop rhythm of rheumatic infection in children is a sign of myocarditis. There is a noteworthy difference in the frequency of gallop rhythm in cases of endo- (myo-) carditis and pancarditis, on the one hand, and cases of an isolated myocarditis, on the other. This is probably due to a greater degree of myocardial damage in the former group. The only sign of heart disease in $^2/_3$ of the cases belonging to the latter group was a transient prolongation of the P—Q time, or a slight enlargement of the left ventricle.

The degree of myocardial damage should, to some extent, be determinable from the röntgenogram of the heart. A more serious myocarditis probably leads to a more marked enlargement of the heart than would a mild myocarditis. Moreover, the experimental examination disclosed a relation between the degree of dilatation and the degree of microscopic changes, although the parallelism was not absolute. A distribution of the material according to the degree of cardiac enlargement will naturally be approximative. Particularly in the milder cases, often only one examination has been performed. Therefore, a transient enlargement may have escaped registration. Furthermore, the examination has not been carried out so as to give quantitative measures of the cardiac enlargement; instead this has been denoted by expressions such as »slight», »moderate», etc. In spite of this, an attempt has been made to determine the frequency of gallop rhythm at different degrees of cardiac enlargement as compared with the frequency of other signs of

TABLE 18.

Frequency of gallop rhythm and other signs of carditis at different degrees of heart enlargement in 80 cases of acute rheumatic infection in children.

Degree of heart enlargement	Total number of cases	Number of cases with				
		Tachycardia ≥ 120	S—T changes	Prolonged P—Q	Pathological murmurs	Gallop rhythm
Distinct	22	6	6	11	15	11
Slight	17	0	1	6	5	4
None	41	0	1	15	2	2

carditis in the electro- and phonocardiogram. The result is presented in Table 18.

It is evident from Table 18 that the gallop rhythm has occurred, above all, in cases with a distinct cardiac enlargement and has been at least as common as other signs of carditis, possibly with the exception of the pathological murmurs. In the cases without a cardiac enlargement, gallop rhythm, changes in the S—T segment and murmurs have been only rarely observed. On the other hand, a prolongation of the P—Q time has been almost as common in these cases as in those with a marked cardiac enlargement. This may possibly indicate, that a prolongation of the conduction time in rheumatic infections need not necessarily be a sign of cardiac damage. BRUENN (1937) and KEITH (1938) have demonstrated how the prolongation may be neutralized in a great number of cases by the injection of atropine and is, therefore, partly due to an increased vagal excitability which is, apparently, characteristic of the rheumatic infection.

The phonocardiographic registration has, as a rule, been performed immediately after admission to the hospital. Subsequently repeated examinations have been made at intervals of varying length, according to the degree of severity of the disease. In over half of the cases of gallop rhythm, this was ascertained at the very first examination together with other signs of carditis (cp. Fig. 15 and Diagram 1). In the remaining

cases it occurred at a later stage, and then either other signs of carditis were found simultaneously (cp. Fig. 16 and Diagram 2) or no changes whatsoever were observed. In some cases, the gallop rhythm again disappeared during the course of the disease and, generally, parallel to the other signs of carditis (cp. Fig. 15). In other cases it remained for a varying length of time after the discharge from hospital, usually for some weeks or months. In 1 case, even 6 years after the acute rheumatic infection, a distinct gallop rhythm was registrable an after-examination revealing a mitral defect. Apart from a pathological configuration, also a moderate enlargement was seen in the röntgenogram. The patient had very insignificant symptoms of cardiac insufficiency and no signs of a myocarditis in the electrocardiogram.

As mentioned previously, the extra sound was a pathologically increased auricular sound in 1 of the 17 cases with a gallop rhythm. In this case the röntgenogram showed an enlargement of chiefly the left ventricle and auricle but no signs of stasis nor of manifest cardiac insufficiency. 10 cases represented third sound gallops. In 1 of them, the nature of the gallop rhythm could not be established during the first weeks of the infection. However, when the pulse frequency fell and the P—Q time became shorter, a typical third sound gallop was recorded in the phonocardiogram. This case showed signs of severe pancarditis. In another case with a third sound gallop, the patient died after 1 week's illness. Autopsy revealed a cardiac enlargement with stasis and peri- as well as myocarditis. All the remaining cases of typical third sound gallop were clinically benign. However, the majority had a distinct cardiac enlargement in the röntgenogram and have later, as the result of the infection, sustained a valvular defect.

Finally, 6 cases were summation gallops. 5 of them showed considerable cardiac enlargement in the röntgenogram and definite signs of decompensation. 3 of them died after a brief period of illness. Autopsy was performed in 1 case, revealing pancarditis with pronounced dilatation of the heart and stasis.

Thus, in the examined cases, the gallop sounds in the severe cases with manifest signs of decompensation were principally

summation sounds, while they in the cases with a more benign course generally represented typical third sounds.

Summary: The examination of children with an acute rheumatic infection has shown that gallop rhythm is not an uncommon symptom in this disease. It is an important sign in as much as it is principally registered in children with definite röntgenological and clinical signs of carditis. In this connection, the summation gallop seems to have a more serious clinical significance than the third sound gallop. The experimental examination disclosed that gallop rhythm in rabbits appeared in connection with a myocardial lesion. This and the clinical examination speaks in favour of the assumption that gallop rhythm in rheumatic infection is a manifestation of myocarditis. Accidentally the present investigation also shows that a prolonged P—Q time, in contrast to gallop rhythm, occurs as often in cases with as in cases without röntgenological and clinical signs of carditis. Therefore, it seems probable that the prolonged P—Q, at least in some cases, is a sign of the general rheumatic infection and not of carditis in particular.

Gallop rhythm in rheumatic defects.

As already mentioned, the gallop rhythm in 6 cases occurred in connection with a valvular defect. These defects all represented mitral lesions. In 5 of them, signs of a combined insufficiency and stenosis were seen to have been present. 3 of the cases had, in all likelihood, been subjected to a relapse of the carditis at the time when the gallop rhythm was ascertained. All these showed cardiac enlargement in the röntgenogram and signs of insufficiency. The rest did not disclose any definite signs of an active carditis and the acute infection dated one or two years back in time. However, they all had more or less distinct signs of cardiac insufficiency, and a cardiac enlargement was observed in the one case submitted to a Röntgen examination. The cases which probably had an active carditis in addition to the valvular defect were all summation gallops, while the others were third sound gallops.

As in the cases of active rheumatic infection, the gallop

rhythm in these children, suffering from a rheumatic valvular defect, was in all probability either a manifestation of myocarditis or of a remaining myocardial damage after the acute infection.

For practical reasons, still another case of rheumatic infection has been referred to this group, although no definite signs of a valvular defect occurred, nor of a carditis. This patient had been subjected to a chorea 4 years previously when the gallop rhythm was first ascertained, as far as is known, without a cardiac complication. No signs of cardiac insufficiency were noted. The electrocardiogram revealed a pronounced prolongation of the conduction time which had, nevertheless, remained practically constant at repeated examinations. No Röntgen examination was performed in this case which had a marked summation gallop. Probably, an acute myocarditis had occurred in connection with the chorea and the gallop rhythm may, perhaps, be interpreted as a remaining sign of this disease.

Gallop rhythm in other infections

Apart from rheumatic carditis, gallop rhythm also occurred in connection with carditis of other origin. In 3 cases the disease had the typical course of a *septic endocarditis*, although the diagnosis could not be verified by blood cultures. All disclosed severe signs of decompensation and considerable cardiac enlargement in the röntgenogram. 1 of the children — in whom a summation gallop had been noted — died. Autopsy was not performed in this case. In another case, the nature of the gallop rhythm could not be established during the earlier phase of the disease. However, as the pulse frequency fell in connection with the patient's improvement, a typical third sound gallop appeared in the phonocardiogram. This was still present a year after the acute illness, while other signs of carditis were no more discernible. In the third case a summation gallop was registered: this was imperceptible at repeated examinations after discharge from the hospital. Now, several years afterwards, the patient no longer displays any signs of cardiac insufficiency whatsoever

and the röntgenogram discloses but a slight hypertrophy of the left half of the heart.

Furthermore, gallop rhythm occurred in 1 case of severe *pancarditis of unknown etiology*. In addition to an auricular sound gallop, pronounced signs of decompensation and a röntgenologically ascertainable enlargement of the heart, including the auricles, were found. This patient died and autopsy revealed considerable peri- and myocarditis but no endocarditis worth mentioning.

In these 4 cases, the gallop rhythm was in all probability of the same significance as in the cases of acute rheumatic infection, i. e. was a sign of myocarditis.

1 case represented a *myocarditis after »influenza»*. This case which had a third sound gallop disclosed no definite signs of cardiac insufficiency and the röntgenogram showed but a slight general enlargement. Electrocardiographic signs of carditis — prolonged conduction time and changed S—T segments — were found. The patient was not subjected to control examinations until several years later when the gallop rhythm as well as the electrocardiographic changes could no longer be registered.

Still another case has been included in this group where a mitral defect was found, probably the result of an earlier, unknown infection. Several years previously this patient had been confined to bed for 3 months due to a cardiac inflammation appearing in connection with an otitis. Considering that a sister of the patient simultaneously suffered from a cardiac inflammation, following a tonsillitis, this was in all likelihood a case of *scarlet fever*. The patient has been examined repeatedly during a period of 5 years and constantly revealed a summation gallop and a P—Q time varying between 0.20 and 0.25 seconds. During this period no symptoms of cardiac insufficiency have occurred and the röntgenogram revealed no cardiac enlargement. The gallop rhythm in this case may probably be conceived as a remaining sign of myocarditis.

Gallop rhythm in congenital heart defects

Gallop rhythm was observed in connection with a congenital defect in 5 cases. 4 of these were clinically benign since there were no symptoms of distinct cardiac insufficiency. However, 1 of them, who suffered from a patent ductus arteriosus, disclosed a certain shortness of breath at exertion. The Röntgen examination showed a moderate enlargement of the left half of the heart. The child was subsequently operated upon but was not subjected to control examinations after the operation. In another case with the same diagnosis, a gallop rhythm was ascertained 4 years after the surgical intervention. However, it may conceivably have been present before but escaped observation. The phonocardiographic registration earlier always took place over the pulmonary artery while the gallop rhythm may have been registrable only over apex. The röntgenogram showed a slight enlargement of the left half of the heart in this case. A third case had a complete heart block, probably due to a septal defect. In a fourth case no detailed diagnosis could be made. In the two last-mentioned cases, the heart revealed moderate enlargement, suggesting a defect. No signs of myocarditis were observed in these cases nor in the two preceding ones. All 4 cases disclosed a third sound gallop. In 1 case, an infant who died only 6 days old, a summation gallop or, possibly, an auricular sound gallop occurred. The autopsy revealed a transposition of the great vessels, hypertrophy of the right half of the heart and a general dilatation. The autopsy report lacks data regarding the condition of the myocardium.

The significance of the gallop rhythm in these cases is unclear. On the whole, it is a rare symptom in the large group of heart diseases formed by the congenital defects. Among the 306 children with this diagnosis treated at the hospital during this examination period, a gallop rhythm occurred only in the forementioned 5 cases. This is noteworthy and does not contradict the conception propounded earlier of gallop rhythm as a sign of myocarditis, since myocarditis is relatively rare in congenital defects. There are, of course, elevations and depressions of the S—T segments to no small extent, above all,

at morbus ceruleus (MANNHEIMER 1940). However, it is by no means unlikely that these changes are generally not signs of a myocardial lesion in the usual sense of the word but, like the pathologically altered initial complexes, rather a manifestation of a congenital damage. It may also be pointed out that a prolonged P—Q very seldom occurs at congenital defects.

Gallop rhythm in anemia

Finally, gallop rhythm has been encountered in 1 case of ulcerative colitis, in 1 case of septic coxitis and in 2 cases of acute leucemia. In all these cases a pronounced anemia was disclosed at the time of the examination, with haemoglobin values, acc. AUTENRIETH, of 23, 38, 34 and 32 per cent and a number of red blood corpuscles per cu. mm of 2.25, 2.48, 1.68 and 1.76 millions. In one of the leucemia cases the röntgenogram revealed a general dilatation of the heart. The other cases were not submitted to a Röntgen examination. In all these cases a pronounced tachycardia of ≥ 120 beats per minute and a summation gallop were found but no electrocardiographic changes.

It is well-known that severe anemia may cause myocardial damage through malnutrition. It therefore seems probable that the gallop rhythm in these cases was a manifestation of a cardiac lesion of this type. This all the more, since it disappeared simultaneously with the anemia in 2 of the cases which have later recovered.

Other cases of gallop rhythm

All the cases of gallop rhythm so far described have been referred to certain well-defined disease groups. As has already been pointed out, in the majority of cases studied here the gallop rhythm has been found in children, who did not disclose symptoms or signs characteristic of any definite disease. On the contrary, they have on the whole appeared to be in good health, have lead a normal life, gone to school and have to a large extent taken part in gymnastics, etc. 10 of the 65 cases

belonging to this group were submitted for examination either because of a certain shortness of breath at exertion, or on account of precordial pain. Troubles of this kind have occurred in another 10 cases, this, however, not being the immediate reason for examination.

As a rule, the physician (in many cases the school doctor) on auscultation has found a systolic murmur, the nature of which has been difficult to determine merely by means of stethoscopy thereupon remitting the child for phonocardiographic registration. In other cases, a tachycardia may have been present, or an extra systole, or some other anomaly, or else the exact reason for the examination has not been given.

The gallop rhythm has only exceptionally been mentioned in the diagnostic report of the remitted patient. This may be due to two specific circumstances. Either the gallop rhythm may have been heard but not attributed any particular significance, or it has escaped observation, the latter probably being the most usual. As already pointed out, the gallop rhythm occurs almost exclusively in the supine position. Since the children have, in all likelihood, in several cases been examined in the erect position, it is easy to understand that the gallop rhythm may have eluded the observer under such conditions. However, even when the supine position has been assumed it is conceivable that the investigator has not listened to the heart sounds sufficiently far out at the apex. As stated earlier, the gallop rhythm is frequently perceived in a limited area over the ictus.

In no instance has diphtheria or rheumatic fever been noted in the case history. 13 of these children have previously been subjected to scarlet fever, 5 of whom fell prey to this disease within a year prior to the examination. 12 cases had suffered from an angina with a high fever, 8 of which less than a year before the examination. Otherwise, only the common infections of the pharynx or the upper air passages or one of the ordinary childhood diseases without complications have been reported in the anamnesis.

As regards the age at which a gallop rhythm was first ascertained, 24 of the children belonging to this group were between 3 and 7 years old, 26 between 8 and 12, and 14

between 13 and 17 years. The fact that the older children are, relatively speaking, fewer than the younger ones may be due to their having been referred for examination to a lesser extent. The work at the heart station has principally concerned children who have not yet reached puberty. Only 1 child was less than 3 years old. Accordingly, the gallop rhythm in this group seems to be a rare occurrence during the first years of life. More than $^2/_3$ were girls and, considering that on the whole an equal number of boys and girls have been examined, it would appear that the gallop rhythm is more common among the latter. However, the difference in distribution between the sexes is not statistically significant, although it is probable.

On account of the absence of tachycardia, it has been possible to determine the nature of the gallop rhythm in the majority of cases included in this group. All of them have been third sound gallops. Only in 6 cases has the nature of the gallop rhythm been unascertainable. These cases have been described as summation gallops, in accordance with the definition adopted above (see page 82).

As already mentioned, the material derives from the years 1939—1945. The examination has generally comprised Röntgen and tolerance tests, apart from the electro- and phonocardiographic registration. However, the tolerance test has only been at disposal since the latter part of 1945. Only since that time röntgen examinations were regularly performed in these clinically benign cases. For this reason the earlier cases have been subjected to an after-examination, inter alia, by means of these methods. Some of them no longer revealed a gallop rhythm at the time of the after-examination.

Changes in the electrocardiogram have occurred in 17 cases and consisted, above all, of a prolongation of the conduction time. This exceeded 0.20 seconds in 9 cases, and equalled 0.19—0.20 seconds in 4. In addition, it often disclosed a considerable variability at the different examinations and, as a rule, did not show any tendency towards a decrease even several years after the first examination. Apart from these disturbances to the P—Q time, changes in the S—T segments and in the initial complexes occurred in 6 cases.

Systolic murmurs were registered in the frequency range of 175—400 cycles per second in almost half of the cases, while in the normal material they were noted but exceptionally in this frequency range. In 4 cases the systolic murmur was recorded up to 500 cycles per second which is pathological.

A Röntgen examination was performed in a total of 48 cases. 6 of these cases showed a pathological configuration of the heart, consisting of an increased bulging of the left ventricular contour which would indicate a certain hypertrophy of the left ventricle. The experimental investigation and the examination on children with acute rheumatic infection both revealed a definite connection between the gallop rhythm and a dilatation of the heart. It is therefore remarkable that a cardiac enlargement was not more often obvious in these 48 cases and when existing, was so insignificant.

In order to find out whether, by means of an objective method, a cardiac enlargement could be ascertained in a greater number of cases, the heart volume was determined in 29 of these patients. The examination was performed with the patient both standing up and lying down. In the supine position, röntgenograms were taken simultaneously in two perpendicular planes (the frontal and the sagittal plane) and the focal distance equalled 100 cm in either direction. In the erect position, the examination similarly included a frontal as well as lateral view with a focal distance of 150 cm, but owing to insufficient technical equipment, the exposures could not be made simultaneously in this position. The heart volume was determined according to KAHLSTORF'S formula modified acc. JONSELL 1939.

In order to obtain a comparable normal material, the heart volume of 61 healthy children in corresponding ages was determined in the same manner. These children were selected from the normal cases described above (cp. page 63). The result is illustrated in Table 19, where the heart volume of normal children in different age-groups in the supine as well as the erect position is demonstrated. It is evident from this table that the heart volume in the examined cases throughout was greater in the supine than in the erect position. The difference is but slight as regards the smaller children, but seems to

TABLE 19.

Heart volume in cm³ in children at different ages in supine and erect position.

Normal cases.

N=number of cases. $M \pm \varepsilon(M)$=mean±standard error of the mean. σ=standard deviation. $D \pm \varepsilon(D)$=difference±standard error of the difference.

Age in years	Supine position			Erect position			$D \pm \varepsilon(D)$
	N	$M \pm \varepsilon(M)$	σ	N	$M \pm \varepsilon(M)$	σ	
4—6	20	274±13	57	20	262±12	55	12±18
9—11	20	446±20	88	19	393±15	67	53±25
14—16	21	810±41	187	21	667±32	146	143±52

become more marked with increasing age. There is, no doubt, a connection between this phenomenon and the difference in pulse frequency in the two positions noted in children after the age of 6—7 years (according to an unpublished investigation made by GUNNAR DAHLBERG). As regards the older children, the difference in heart volume is statistically probable, which is in conformity with earlier findings by NYLIN (1934) on adults.

It has been stated above (cp. page 77 and 83) that the physiological third sound as well as the third sound gallop in small children has not rarely been heard and registered even in the erect position while in older children it occurs almost exclusively in the supine position. This may probably be explained by the fact that the heart volume in the small children is practically the same in either position.

In examinations on adults, the heart volume has been correlated to various factors such as the body weight, length and surface, the best correlation being to body weight and body surface (BIÖRCK 1944). In infants, the heart volume shows as good a correlation to the length of the body and the age as to the body weight (AXÉN and LIND, 1945). Apparently, no similar examinations were performed on older children.

In the normal cases examined in the present work, the heart volume has been correlated to body weight and body surface,

TABLE 20.

Correlation between heart volume and body weight and body surface in children.

Normal cases.

N=number of cases. $r \pm \varepsilon(r)$=correlation coefficient±standard error of the correlation coefficient.

Correlation	Body weight $\leq$ 45.0 Kg Body surface $\leq$ 1.40 m²		Body weight $\geq$ 45.0 Kg Body surface $\geq$ 1.40 m²	
	N	$r \pm \varepsilon(r)$	N	$r \pm \varepsilon(r)$
Heart volume in supine position and body weight	41	+0.91±0.026	20	+0.54±0.16
Heart volume in erect position and body weight	40	+0.89±0.032	20	+0.67±0.12
Heart volume in supine position and body surface	40	+0.93±0.022	21	+0.70±0.11
Heart volume in erect position and body surface	39	+0.90±0.032	21	+0.71±0.11

the latter being calculated acc. DUBOIS' formula. The material has been divided into 2 groups, the limit between them being set at 45.0 Kg of body weight and 1.40 m² of body surface, which on the whole corresponds to the beginning of puberty. The correlation coefficients are shown in Table 20.

As may be seen from Table 20, the heart volume at a body weight of $\leq$ 45.0 Kg and a body surface of $\leq$ 1.40 m² shows a very good correlation to both these factors. This would indicate a practically rectilinear relationship. The correlation is not so good when the body weight and the body surface exceed these figures. It is conceivable that certain sex differences may be manifest after puberty. However, it has not been possible to establish such a difference on the basis of the small number of determinations made here.

The heart volume per Kg body weight and per m² body surface has been determined in 41 normal cases and 29 cases of gallop rhythm. Because of the circumstances mentioned in

TABLE 21.

Heart volume per Kg body weight and per m² body surface in normal children and children with gallop rhythm. Body weight ≤ 45.0 Kg and body surface ≤ 1.40 m².

N=number of cases. $M \pm \varepsilon(M)$=mean ± standard error of the mean. σ=standard deviation. $D \pm \varepsilon(D)$=difference ± standard error of the difference.

Position	Normal cases			Cases with gallop rhythm			$D \pm \varepsilon(D)$
	N	$M \pm \varepsilon(M)$	σ	N	$M \pm \varepsilon(M)$	σ	
Heart volume per Kg body weight							
Supine	41	12.8±0.21	1.4	29	12.7±0.31	1.7	0.1± 0.37
Erect	40	11.9±0.21	1.3	29	11.5±0.32	1.7	0.4± 0.38
Heart volume per m² body surface							
Supine	40	354.7±7.6	48.0	26	354.0±11.6	59.3	0.7±13.9
Erect	39	326.3±6.3	39.1	26	322.9± 9.2	46.7	3.4±11.2

the preceding paragraph, only children with a body weight of ≤ 45.0 Kg and a body surface of ≤ 1.40 m² have been included. The values obtained have been subjected to statistical analysis with regard to the mean and the range of variation. The result of the examination is set forth in Table 21.

Table 21 shows that in the cases of gallop rhythm the mean heart volume per Kg of body weight as well as per m² of body surface did not differ from the corresponding values in the normal cases. This applies both to the erect and the supine position. In all cases but one or two, the heart volume lay within the normal range of variation.

Hypoxia tolerance tests of the heart were performed in 58 cases, according to the method described by MANNHEIMER (1946). Thus, the children for 10 minutes breathed a mixture containing 9 per cent of oxygen. Electro- and phonocardiographic registrations were made, during the tolerance test as well as before and after. MANNHEIMER showed that normal children react negatively to this tolerance test while children suffering from various heart diseases, such as active rheumatic carditis, certain congenital defects, etc., disclose changes in the electrocar-

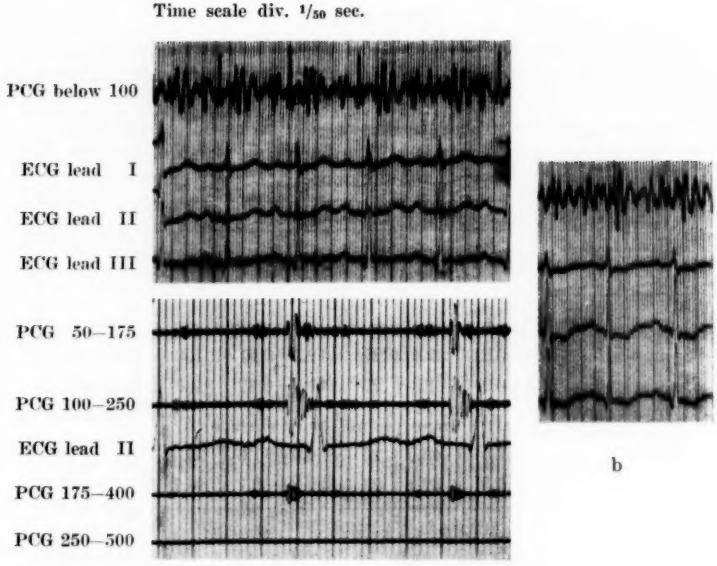


Fig. 17 a and b. ECG 1384/45. A girl, aged 4 years. No distinct morbid symptoms. With the exception of a bronchial catarrh a few months previously, always in good health. Normal heart röntgenogram. (a) ECG and PCG before hypoxia tolefance test: P—Q time rather long for her age, i. e. 0.17 seconds. Otherwise normal ECG. Pronounced summation gallop with the extra sound registered right up to the frequency range of 250—500 cycles per second. The amplitude is everywhere larger than that of the first and second sound. Systolic murmur of a fairly high frequency of 50—400 cycles per second. (b) ECG during hypoxia tolerance test (9 per cent oxygen-gas for 10 minutes): Distinct depressions of the S—T segment in all leads.

diogram during the test consisting mainly of changes in the S—T segments in one or several leads.

In 12 of the 58 examined cases, a gallop rhythm had occurred earlier although it could not be registered at the time of the tolerance test. The remaining 46 cases had a distinct gallop rhythm at the time of the examination. In the first-mentioned group, 1 reacted positively to the tolerance test while in the

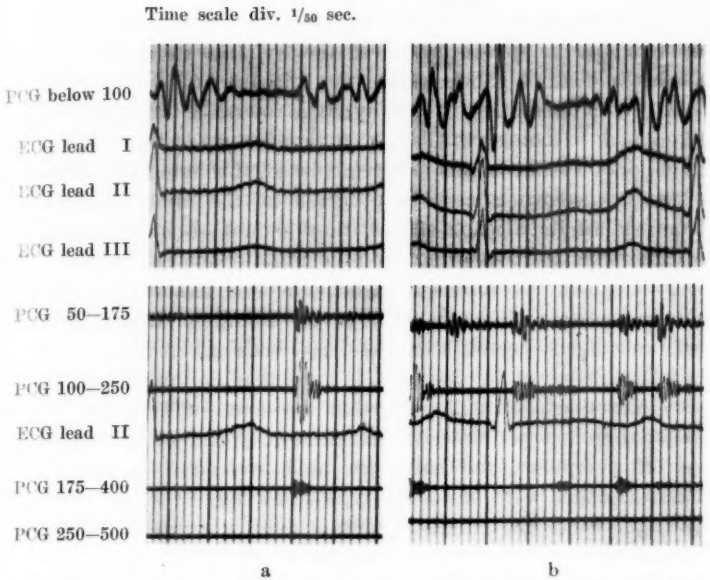


Fig. 18 a and b. ECG 189/44. A boy, aged 13 years. Two years before, a gallop rhythm was registered without other signs of cardiac disease. (a) ECG and PCG before hypoxia tolerance test: Normal ECG. Somewhat pronounced third heart sound, though within physiological limits. Faint systolic murmur of 50—250 cycles per second. (b) ECG and PCG during hypoxia tolerance test (9 per cent of oxygen-gas for 10 minutes): No changes in the ECG. Gallop rhythm with the third sound registered up to the frequency range of 175—400 cycles per second. The systolic murmur stronger and of higher frequency.

last-mentioned group 23 showed a pathological reaction. The occurrence of pathological changes in the röntgenogram and in the electrocardiogram have not been decisive with regard to the result of the test, i. e. they have been noticed equally often among the cases who reacted positively as among those with a negative reaction. In 16 cases a positive reaction to the tolerance test was the only pathological finding (see Fig. 17).

In 2 of the 12 cases where a previous gallop rhythm had disappeared at the time of examination, this reappeared during the hypoxia tolerance test (see Fig. 18).

In addition, a marked accentuation of the gallop sound occurred under the influence of the hypoxia in a few cases having a distinct gallop before the tolerance test.

A positive reaction to the hypoxia tolerance test is, in all likelihood, pathological. The exact significance of this symptom in childhood still remains to be elucidated, this being the subject of an investigation now in progress at the clinic. Judging from the findings made so far, they should be interpreted as a sign of a manifest or latent myocardial damage.

By means of the forementioned methods of examination, viz. electrocardiogram, phonocardiogram, Röntgen and hypoxia tolerance tests, certain changes have been ascertained in the hearts of 39 out of 65 cases, i. e. in more than half of the children who had disclosed gallop rhythm but had, nevertheless, not suffered from any disease insomuch as they had not shown any morbid symptoms and had been able to lead a normal life. These changes were of such a type as to suggest a myocardial damage. Since the gallop rhythm in acute rheumatic infection is, in all likelihood, a sign of myocarditis, it may be assumed to be of a similar significance in these mild cases, i. e. to be a manifestation of a myocardial lesion.

Discussion

The result of the experimental investigation, revealing the close relationship between gallop rhythm and cardiac dilatation, favours the conception that the third sound gallop at least, is due to a diminished tone of the cardiac muscle caused by myocardial damage. Furthermore, in children with active rheumatic carditis the gallop rhythm was principally observed in cases, where a distinct cardiac enlargement was found. Thus, it seems probable that the mechanism of origin was the same in these cases.

It is noteworthy that a gallop rhythm, as marked as the one observed in certain severe cases of carditis, may occur in children who do not disclose any definite clinical signs of disease and where the Röntgen examination reveals no cardiac enlargement. However, it seems probable that the gallop rhythm

in these cases too, is a manifestation of a decreased muscular tone, although this is not so pronounced as to give rise to a dilatation. Either a fairly mild form of myocardial damage may be conceived in these cases, or it may perhaps be the last trace af a previous, more pronounced myocarditis.

Such a decreased tone may possibly also be caused by other factors. The significance of the vegetative nervous system, above all that of the vagus, with regard to the appearance of the physiological third sound has often been stressed in the literature and has also been experimentally proved by MELIK-GÜLNASARIAN (1932). LUISADA and MAUTNER (1944) claim to have produced even a gallop rhythm by experimental means. This was achieved by injection of certain drugs principally having a stimulating effect on the vagus. It is, therefore, not at all unlikely that a gallop rhythm also in children may be caused merely by an increased vagal tone, in which case it might be possible to make it disappear by means of substances with an opposite effect. Preliminary investigations show that a gallop rhythm may sometimes disappear completely after the injection of atropine. However, this often happened also in cases with signs of myocardial damage, and it would probably not be possible to distinguish between gallop rhythms of different etiology by these methods.

A gallop rhythm, in accordance with the definition made in the present investigation, has not been noted in any of the normal cases. However, this may be purely a matter of chance considering the relatively small number of examined children. For the present, the possibility of a gallop rhythm occurring in normal cases cannot be completely excluded.

Thus, even though it cannot definitely be propounded that gallop rhythm in children under all circumstances is a sign of myocarditis, this is undoubtedly the rule. Changes in the heart of various kinds, which should probably be interpreted as signs of a myocardial damage, have been observed to such a great extent in connection with gallop rhythm as to justify a close examination of each single case with this particular sign, even though the child otherwise fails to show any definite symptoms of disease.

Results of the investigation

From the survey of the literature it will be seen that the gallop rhythm in adults has been subjected to detailed clinical analysis, having been found above all in elderly persons with degenerative heart diseases. As a rule, it has been connected with a pathological reduction in tone of the ventricular wall, due to a myocardial damage, whether infectious, toxic or otherwise. However, as far as is known, experimental examinations have not been performed earlier in order to demonstrate such a relationship.

Very little notice has been paid to this symptom in children. A gallop rhythm has been described in connection with acute infectious diseases and has then been regarded as a sign of a weak heart. Since audible extra sounds are to some extent a normal occurrence in children, it may be questioned whether a true gallop rhythm was actually present in all these cases.

It has generally been considered impossible to distinguish between gallop sounds and the corresponding physiological extra sounds whether by means of auscultation, or by the use of the phonocardiographic registration methods adopted earlier. Therefore, the term gallop rhythm has usually been employed in cases manifesting distinct signs of cardiac disease, while the extra sound was regarded as physiological when the heart disclosed normal activity.

This is obviously an unsatisfactory definition. The gallop rhythm must be objectively defined to be useful as a diagnostic aid. By means of calibrated phonocardiography, MANNHEIMER (1940) showed, that distinctly audible gallop sounds have a higher frequency and a larger amplitude than the normal third sound and the auricular sound. Thus, by this method it should be possible to obtain an objective definition of the gallop rhythm.

Accordingly, it has been considered justified to perform an investigation on children on a broader scale.

The method of phonocardiographic registration, dealt with in Chapter II, is adopted in the present investigation according to the description given by MANNHEIMER (1940). The tonal scale is divided into 6 frequency ranges, viz., < 100, 50—175, 100—250, 175—400, 250—500 and 500—1000 cycles per second. By simultaneous registration of these frequency ranges, a detailed study is possible of the manner in which fundamental sounds and overtones combine to produce the various sound phenomena. In this way, an expression of their frequency is obtained. The calibration permits a quantitative measurement of the amplitudes in the various frequency ranges in dynes per cm^2 and the method is, accordingly, reproducible.

A myocardial damage has been obtained in 23 out of 30 rabbits (see Chapter III) by means of a method elaborated by FLEISHER and LOEB, consisting of an intravenous injection of coffein and adrenalin. It was then studied by electrocardiography, phonocardiography, Röntgen and a patho-anatomical examination. After the injection a gallop rhythm occurred in 12 of the 30 animals. A third sound gallop was ascertained in all cases where the nature of the gallop rhythm could be determined. A Röntgen examination was performed in 24 cases, revealing a distinct dilatation in 13. A dilatation as well as a gallop rhythm occurred exclusively among the cases where the patho-anatomical examination disclosed clear signs of myocardial damage. Among 10 cases of gallop rhythm where röntgenograms were taken, a dilatation was noted in 9. This indicates a close relationship between these signs.

The electrocardiographic and röntgenological changes, like the gallop rhythm, generally set in on the 4th to 6th day after the injection. This corresponds to the time when distinct microscopical changes begin to appear in the myocardium, according to the observations reported by earlier investigators.

The close relationship between gallop rhythm and dilatation, as shown in the experimental investigation, favours the conten-

tion that the gallop rhythm is due to a pathologically reduced tone of the cardiac muscle caused by the myocardial damage. Thus, the conception of the mechanism of origin of, above all, the third sound gallop, arrived at by clinical observations on adults, derives strong support from this investigation.

The normal phonocardiogram has been studied in 150 children and young persons aged from 3—17 years (see Chapters IV and V), special regard being paid to the third sound and the auricular sound. In accordance with the findings of earlier authors, these sound phenomena were registered to a large extent, i. e. the third sound in 78.0 ± 3.4 per cent and the auricular sound in 50.0 ± 4.1 per cent. Both represent sound phenomena of very low frequency, in contrast to the first and second sounds, the auricular sound being recorded only in the frequency range of < 100 cycles per second, while the third sound was recorded in 8.0 ± 2.2 per cent also in the frequency range of 50—175 cycles per second.

The amplitudes have been calculated statistically with regard to the mean and the range of variation.

On the basis of this normal material, *the gallop rhythm in children* has been defined as follows:

Third sound gallop is present

when the third heart sound is registered in the frequency range of 100—250 cycles per second or higher, or

when the amplitude of the third sound in the frequency range of $<$ 100 cycles per second exceeds 50—60 dynes per cm², and in the frequency range of 50—175 cycles per second 10—15 dynes per cm².

Auricular sound gallop is present

when the auricular sound is recorded in the frequency range of 50—175 cycles per second or higher, or

when the amplitude of the auricular sound in the frequency range of $<$ 100 cycles per second exceeds 30—40 dynes per cm².

This definition cannot be generally applied as it is dependent on the particular frequency ranges selected. At a different

distribution of the frequency ranges other values of the amplitudes would be obtained. Nor does it claim exactness. The normal material subjected to examination is relatively restricted. If a greater number of children had been examined, the values of the range of variation would probably have deviated somewhat. However, by this definition it is possible to make a distinction between physiological and pathological extra sounds, which could not be done previously.

During the period of 1939—1945, about 5000 children were examined at the Heart Station of the Crown Princess Lovisa's Children's Hospital by means of calibrated phonocardiography. Among them, 104 cases of gallop rhythm occurred. These were submitted to a close analysis, as described in Chapter VI, with particular regard to the occurrence of morbid symptoms and signs.

Among these 104 cases, a third sound gallop was noted in 78 and an auricular sound gallop in 2. Criteria of both a third sound and an auricular sound were observed in 24 cases, the term summation gallop being applied here in accordance with current nomenclature.

Like the corresponding physiological extra sounds, the gallop sounds were recorded with the largest amplitude at the apex. Earlier investigations have been confirmed in this respect.

The gallop rhythm, like the normal third and auricular sounds, is best or exclusively registered with the subject lying down. This is also statistically proved by an examination of 39 cases of third sound gallop and is probably due to altered physiological conditions in the supine as compared to the erect position.

The gallop rhythm was often observed in connection with a rheumatic disease. It was registered in 17 out of 88 cases of polyarthritis acuta and chorea minor, treated at the hospital during the above-mentioned period of observation. Gallop rhythm is, accordingly, not an uncommon symptom in acute rheumatic infection. These 17 cases all disclosed distinct röntgenological and clinical signs of carditis. This — viewed against the background of the experimental investigation — favours the assumption of the gallop rhythm being a sign of myocarditis in

these instances. The summation gallop has been found to be a more serious clinical sign than the third sound gallop insofar as the former has occurred principally in the more severe cases. In certain instances, the gallop rhythm remained for a varying length of time after the discharge from hospital. In 1 case it was still registered 6 years after the acute infection.

The gallop rhythm was also observed in connection with other infections such as, for instance, septic carditis. In these cases, the gallop rhythm may probably be attributed the same significance as in rheumatic infection.

The gallop rhythm was reported in only 5 out of 306 cases of congenital heart defect. It is evidently a rare symptom in this type of cardiac disease.

In a few cases, the gallop rhythm was ascertained in certain other diseases which had one feature in common, i. e. a pronounced anemia at the time of the examination. In these cases the gallop rhythm was probably a sign of a myocardial damage owing to mal-nutrition. This damage may be regarded as reversible in two cases where the gallop rhythm disappeared when the anemia was cured.

In 65 of the 104 cases examined here, the gallop rhythm was noted in children who did not disclose any characteristic signs of disease and were able to lead a normal life. 13 of them had, however, suffered from scarlet fever and another 12 from angina tonsillaris, as a rule, during the last two years preceding the examination. 20 of the children complained of slight symptoms such as palpitations or a mild degree of breathlessness on effort. By means of special investigation methods, such as electrocardiography, phonocardiography, Röntgen and hypoxia tolerance tests, certain pathological changes in the heart have been ascertained in a little more than half of the cases. The nature of these changes seemed to indicate a latent myocarditis or some other myocardial damage.

Determinations of the heart volume have been performed in 29 cases belonging to this group. In all but 2 the heart volume lay within the normal range of variation. This is remarkable considering that the experimental investigation as well as the examination of children with an acute rheumatic infection dis-

closed a distinct relationship between gallop rhythm and cardiac enlargement. Apparently, the gallop rhythm may also be a manifestation of a fairly mild myocardial damage.

However, in several cases no signs of cardiac disease whatsoever are noticed, apart from the gallop rhythm. A cardiac disease of some kind may, conceivably, have occurred earlier, the gallop rhythm being a sign of a remaining myocardial damage after the disappearance of the other symptoms. It is also possible that the gallop rhythm may be the only sign of a myocardial affection, infectious, toxic or otherwise.

Other etiological factors cannot, however, be excluded. The effect of the vegetative nervous system, above all that of the vagus, on the occurrence of the physiological third sound has often been propounded earlier, and even experimentally demonstrated. An increased vagal tone may, perhaps, involve a reduced diastolic tone of the cardiac muscle to such an extent as to produce a gallop rhythm. Finally, the possibility of the gallop rhythm occurring as a rare phenomenon in completely healthy individuals cannot be definitely excluded.

Thus, though the gallop rhythm cannot in every case be proved to be a sign of myocarditis or of myocardial damage, pathological cardiac changes are, nevertheless, so frequently found in connection with gallop rhythm that more attention than hitherto should be paid to this symptom in children.

Literature

The abbreviations are in accordance with the Quarterly Cumulative Index Medicus.

Axén, O., & Lind, J., Roentgenologic determination of heart volume in infants. A preliminary report. Acta Paediat. *32*: 84, 1945.

Barié, E., Traité pratique des maladies du cœur et de l'aorte. Paris, Vigot Frères, 1912.

Barkesdale, E. H., The effect of lowering blood pressure on myocarditis caused by epinephrin hydrochloride and caffeine in the rabbit. Arch. Path. & Lab. Med. *3*: 658, 1927.

Battro, A., Braun-Menendez, E., & Orías, O., Asincronismo de la contracción ventricular en el bloqueo de rama. ... Rev. argent. de cardiol. *3*: 325, 1936. Ref. to by Orías, O., & Braun-Menendez, E., 1939.

— El ritmo de galope. Rev. argent. de cardiol. *1*: 117, 1934—35.

Becke, W. G., Johnson, S., & Harris, H. S., An electrocardiographic study of experimental heart disease. Am. Heart J. *2*: 302, 1926—27.

Benjamins, C. E., Über die Untersuchung des Herzens von der Speiseröhre aus. Arch. f. d. ges. Physiol. *158*: 125, 1914.

Biörck, G., On the relationship between the heart volume and various physical factors. Acta radiol. *25*: 371, 1944.

Blacher, W., Galopprhytmus und Extrasystolen bei der diphterischen Myocarditis. Jahrb. f. Kinderh. *79*: 166, 1914.

Bouillaud, J., ref. to by Potain, C., 1900.

Bourdet, E., Yacoël, J., & Giroux, R., Les modifications de l'électrocardiogramme dans certains cas de bruit de galop. Arch. d. mal. du cœur *16*: 205, 1923.

Boyer, N., Studies on the third heart sound. Am. Heart J. *23*: 797, 1942.

Bramwell, C., Sounds and murmurs produced by auricular systole. Quart. J. Med. *4*: 139, 1935 (a).

— Gallop rhythm. Ibid. *4*: 149, 1935 (b).

Braun-Menendez, E., & Orías, O., Estudio fonocardiográfico en cien adultos jóvenes. Rev. argent. de cardiol. *1*: 101, 1934—35.

— & Solari, L.-A., Étude des bruits auriculaires dans le bloc auriculo-ventriculaire expérimental. Compt. rend. Soc. de biol. *123*: 502, 1936.

Bridgman, E. W., Notes on a normal, præsystolic sound. Arch. Int. Med. *14*: 475, 1914.

— Observations on the third heart sound. Heart *6*: 41, 1915.

Bruenn, H., The mechanism of impaired auriculoventricular conduction in acute rheumatic fever. Am. Heart J. *13*: 413, 1937.

Caeiro, A., & Orías, O., The phonocardiogram registered in special areas of auscultation. Rev. argent. de cardiol. *4*: 71, 1937. — Author's review in Am. Heart. J. *15*: 372, 1938.

Calo, A., Atlas de phonocardiographie clinique. Paris, Masson et Cie. 1938.

Carlgren, L.-E., Experimentally produced gallop rhythm. A preliminary report. Acta Paediat. *32*: 164, 1945.

Charcelay, A., Arch. génér. de méd. *3*: 393, 1838, ref. to by Holt, E., et al.

Chauveau, H., Étude cardiographique sur le mécanisme du bruit de galop. Thèse de Paris 1902.

Christian, H. A., Smith, R. M., & Walker, I. C., Experimental myocarditis produced by spartein sulphate and adrenalin chlorid. Arch. Int. Med. *8*: 515, 1911.

Clendinning, J., (1840), Brit. Ass. Report on Heart Sounds. Ref. to by O'Farell, P. T., 1939.

Cloetta, M., Über den Einfluss der chronischen Digitalisbehandlung auf das normale und pathologische Herz. Arch. f. exper. Path. u. Pharmakol. *59*: 209, 1908.

Cossio, P., & Braun-Menendez, E., Desdoblamiento fisiológico de los ruidos del corazón. Rev. argent. de cardiol. *2*: 149, 1935.

— & Lascales, M., Premier bruit du cœur et bruit auriculaire. Arch. d. mal. du cœur *29*: 138, 1936.

— & Orías, O., Chasquido de apertura de la mitral. Rev. argent. de cardiol. *1*: 451, 1935.

Dean, A. L., The movements of the mitral cusps in relation to the cardiac cycle. Am. J. Physiol. *40*: 206, 1916.

Decherd, G., Herrman, G., & Erhard, P., Effect of myocardial destructive agents on creatine contents of rabbit's heart. Proc. Soc. Exper. Biol. & Med. *33*: 519, 1936.

Duchosal, P., A study of gallop rhythm by a combination of phonocardiographic and electrocardiographic methods. Am. Heart J. *7*: 613, 1931—32.

— Nouvelles recherches graphiques sur le bruit de galop. Arch. d. mal. du cœur *28*: 345, 1935.

Duroziez, P., Essai sur les maladies du cœur. Du rythme pathognomonique du rétrécissement mitral. Arch. génér. de méd. *5*: *20*: 385, 1862. Ref. to by Frost, J., 1944, et al.

Einthoven, W., Ein dritter Herzton. Arch. f. d. ges. Physiol. *120*: 31. 1907.

Evans, W., Triple heart rhythm. Brit. Heart J. *5*: 205, 1943.

Exchaquet, Th., D'un phénomène stéthoscopique propre à certaines formes d'hypertrophie simple du cœur. Thèse de Paris 1875. Ref. to by Potain, C., 1894.

FAZEKAS, E., Galopprhythmus in Pubertäts- und jugendlichen Alter. Ztschr. f. Kreislaufforsch. *34*: 410, 1942 (a).

— »Physiologischer» dritter Ton und Galopprhythmus. Ztschr. f. Kreislaufforsch. *34*: 543, 1942 (b).

FLEISHER, M., & LOEB, L., Experimental myocarditis, Arch. Int. Med. *3*: 78 1909.

FOGELSON, L. I., Der Galopprhythmus. Klinisch-experimentelle Untersuchung. Ztschr. f. klin. Med. *119*: 205, 1931—32.

— Die Bedeutung der Stärke der Herztöne, besonders des ersten, in der Beurteilung des Zustandes des Myokards. Ztschr. f. Kreislaufforsch. *25*: 654, 1933.

FRIEDLÄNDER, A., Acute rheumatic infection in children. Clinical studies with special reference to the initial phase and the valuation of the cardiologic and hematologic signs. Acta pædiat. *32*: 207, 1945.

— & MANNHEIMER, E., Några drag ur den reumatiska infektionens klinik i barnaåldern med särskild hänsyn till tidigdiagnosen och värderingen av de kardiologiska fynden. Nord. Med. *24*: 2218, 1944.

FROST, J., Galoprytme. En klinisk-fonokardiografisk undersögelse. [Akad. Afh.] Khvn, Ejnar Munksgaard, 1944.

FRAENTZEL, O., Über Galopprythmus am Herzen. Ztschr. f. klin. Med. *3*: 491, 1881.

GALLAVARDIN, L., Bruit de dédoublement mitral et troisième bruit de cœur. Arch. d. mal. du cœur *5*: 776, 1912.

— Pathogénie du galop diastolique; ses rapports avec les troubles de conductibilité auriculo-ventriculaire et l'altération de la contraction ventriculaire. Ibid. *13*: 410, 1920.

GARVIN, C. F., Gallop rhythm — incidence and influence of age, race and sex. Am. J. M. Sc. *205*: 814, 1943. — Author's review in Am. Heart J. *27*: 131, 1944.

GIBSON, A. G., The significance of a hitherto undescribed wave in the jugular pulse. Lancet 1907: 2, 1380.

GRUBER, C. M., OLCH, I. Y., & BLADES, B., Myocarditis produced experimentally in rabbits by drugs. J. Pharmacol. & Exper. Therap. *49*: 306. 1933.

GUBERGRITZ, M. M., Vom dritten normalen Herzton. Ztschr. f. klin. Med. *102*: 109, 1925—26.

— Über gespaltene und sogenannte überzählige Herztöne. Ztschr. f. Kreislaufforsch. *21*: 65, 1929.

HAMILTON, F., The significance of gallop rhythm. Canad. M. A. J. *44*: 260. 1941.

HENDERSON, Y., & JOHNSON, F. E., Two modes of closure of the heart valves. Heart *4*: 69, 1912—13.

HIRSCHFELDER, A. D., Some variations in the form of the venous pulse. A preliminary report. Bull. Johns Hopkins Hosp. *18*: 265, 1907.

HOLT, E., Gallop rhythm. Am. Heart J. *2*: 453, 1926—27.

HÜRTHLE, K., Beiträge zur Hämodynamik. X. Über die mechanische Registrierung der Herztöne. Arch. f. d. ges. Physiol. *60*: 263, 1895.

JOHNSON, S., & SIEBERT, W. J., Experimental myocarditis. Am. Heart J. *3*: 279, 1927—28.

JONSELL, S., A method for the determination of the heart size by teleroentgenography. (A heart volume index.) Acta radiol. *20*: 325, 1939.

KAHN, R. H., Die Lage der Herztöne im Elektrokardiogramme. Arch. f. d. ges. Physiol. *133*: 597, 1910.

KEITH, J. D., Overstimulation of the vagus nerve in rheumatic fever. Quart. J. Med. *31*: 29, 1938.

KREHL, S. L., Über den Herzmuskelton. Arch. f. (Anat. u.) Physiol. *13*: 253, 1889. Ref. to by Hürthle, K., 1895.

KRIEGE, H., & SCHMALL, B., Über den Galopprhythmus des Herzens. Ztschr. f. klin. Med. *18*: 261, 1891.

LAUBRY, C., & MOUGEOT, A., Pathogénie et signification des bruits de galop mésodiastolique et protodiastolique. Bull. et mém. Soc. méd. d. hôp. de Paris *45*: 1623, 1921.

— & PEZZI, C., Les syndromes cardiaques. Les rythmes de galop. Paris, G. Doin & Cie, 1926. Ref. to by Orías, O., & Braun-Menendez, E., 1939, Frost, J., 1944, et al.

— & ROUTIER, D., Bruit de galop et rythme à quatre temps. Bull. et mém. Soc. méd. d. hôp. de Paris *49*: 1281, 1925.

— ROUTIER, D., & LARGEAU, R., L'insuffisance ventriculaire droite. Presse méd. *32*: 681, 1924.

LEONHARDT, W., Über den dritten Herzton und das kindliche Herzschallbild. Ztschr. f. d. ges. exper. Med. *84*: 470, 1932.

LEVINE, H. D., Spontaneous changes in the normal rabbit electrocardiogram. Am. Heart J. *24*: 209, 1942.

LEWIS, J. K., Nature and significance of heart sounds and of apex impulses in bundle branch block. Arch. Int. Med. *53*: 741, 1934.

LEWIS, T., Lectures on the heart. New York & London 1915. Ref. to by Fogelson, L. I., 1933.

LEYDEN, E., Über die Herzaffectionen bei der Diphterie. Ztschr. f. klin. Med. *4*: 334, 1882.

LIAN, C., MINOT, G., & WELTI, J. J., Phonocardiographie, auscultation collective (acoustique—technique—clinique). Paris, Masson & Cie, 1941.

— & RACINE, M., La phonocardiographie. III. Les rythmes à trois temps. Ann. de méd. *34*: 157, 1933.

— & WELTI, J. J., Le dédoublement du premier bruit; le premier bruit à précession auriculaire et le galop présystolique retardé. Arch. d. mal. du cœur *31*: 408, 1938.

LUISADA, A. A., & MAUTNER, H., Experimental studies on functional murmurs and extra-sounds of the heart. Exper. Med. & Surg. *1*: 282, 1943.

McKee, M. H., Heart sounds in normal children. Am. Heart J. *16*: 79, 1938 (a).

— Heart sounds and murmurs in children with rheumatic heart disease. Ibid. *16*: 88, 1938 (b).

Macleod, A. G., Wilson, F. N., & Barker, P. S., Observations on heart sounds with particular reference to gallop rhythm and sounds of auricular origin. Proc. Soc. Exper. Biol. & Med. *29*: 1009, 1931—32.

Mannheimer, E., Calibrated phonocardiography and electrocardiography. A clinical-statistical study of normal children and children with congenital heart disease. Acta pediat. vol. XXVIII, Supplementum II. 1940.

— Kalibrerad fonokardiografi. Nord. Med. *12*: 3617, 1941.

— The interpretation and significance of the various types of gallop rhythm. Acta med. Scandinav. *111*: 442, 1942.

— The hypoxia tolerance test of the heart in children. J. Pediat. Sept. 1946.

Margolies, A., & Wolferth, C. C., The opening snap (»claquement d'ouverture de la mitrale») in mitral stenosis, its characteristics, mechanism of production and diagnostic importance. Am. Heart J. *7*: 443, 1932.

Melik-Gülnasarian, E. A., Über den Tonus des Herzmuskels und den dritten Herzton. Klinisch-experimentelle Untersuchung. Ztschr. f. Kreislaufforsch. *24*: 433, 1932.

Mond, H., & Oppenheimer, E. T., Gallop rhythm in hypertension. Arch. Int. Med. *43*: 166, 1929.

Mozer, J. J., & Duchosal, P., Étude électro-phonocardiographique du rythme du galop. Arch. d. mal. du cœur *23*: 65, 1930.

Müller, F., Über Galopprhythmus des Herzens. München. Med. Wchnschr. *53*: 785, 1906: 1.

Nyboer, J., Non-induced cardiopathic disease in a rabbit — electrocardiographic and pathologic study. Proc. Soc. Exper. Biol. & Med. *44*: 435. 1940.

— Spence, M. J., & MacNeal, W. J., Role of heart sounds in diagnosis of experimental endocarditis lenta in rabbits before death. Ibid. *46*: 545, 1941.

Nylin, G., The relation between heart volume and stroke volume in recumbent and erect positions. Scand. Arch. Physiol. *69*: 237, 1934.

Obrastzow, W. P., Über die verdoppelten und akzessorischen Herztöne bei unmittelbarer Auskultation des Herzens. Ztschr. f. klin. Med. *57*: 70, 1905.

— Vom 3. Herzton. Vortrag in Kiewer Arztverein 1919. Ref. to by Steinberg, L. D., 1925.

O'Farell, P. T., What is gallop rhythm? Irish J. M. Sc. Sér. *6*: 729, 1939.

Ohm, R., Der sogen. dritte Herzton und seine Beziehungen zur diastolischen Kammerfüllung. Berl. klin. Wchnschr. *58*: 600, 1921.

Orías, O., & Braun-Menendez, E., The heart sounds in normal and pathological conditions. London, New York & Toronto 1939. Oxf. Univ. Press.

Pereira, J. C., Estudios fonocardiograficos y hemodinamicos en embarazadas y fetos en los últimos tiempos del embarazo normal. Tesis de doctorado en medicina. Buenos Aires, Sebastián de Amorrortu e Hijos, 1939.

Pezzi, C., Recherches graphiques sur le bruit de galop. Compt. rend. Soc. de biol. *76*: 705, 1914.

Potain, C., Du rythme cardiaque appelé bruit de galop; de son mécanisme et de sa valeur séméiologique. Bull. et mém. Soc. de méd. de Paris *12*: 137, 1875. Ref. to by Orías, O., & Braun-Menendez, E., 1939, et al.

— Clinique médicale de la charité. Leçons et mémoires. Paris, Masson & Cie, 1894.

— Les bruits de galop. Semaine méd. *20*: 175, 1900.

Robinson, G. C., Gallop rhythm of the heart. Am. J. M. Sc. *135*: 670, 1908.

Rouches, F.-J.-M., Du claquement d'ouverture de la mitrale. Thèse de Paris 1888. Ref. to by Frost, J., 1944, et al.

Routier, D., & van Bogaert, A., Contribution à l'étude clinique du bruit de galop. I. Le galop présystolique tactile. Arch. d. mal. du cœur *27*: 389, 1934 (a).

— Contribution à l'étude clinique du bruit de galop. III. Le troisième bruit physiologique. Le galop protodiastolique. Ibid. *27*: 588, 1934 (b).

— Die Störungen im Ekg beim Galopprhythmus. Ztschr. f. Kreislaufforsch. *26*: 604, 1934 (c).

Routier, D., & van Heerswynghels, J., À propos du bruit de galop. Arch. d. mal. du cœur *28*: 629, 1935.

Schrumpf, P., Die Spaltung resp. Verdoppelung der Herztöne. Deutsches Arch. f. klin. Med. *126*: 73, 1918.

Schütz, E., Physiologie der Herztöne. Ergebn. d. Physiol. *35*: 632, 1933.

Segura, A. S., Registro e interpretación de la actividad cardiovascular en el lactante normal. Rev. argent. de cardiol. *3*: 3, 1936. Ref. to by Orías, O., & Braun-Menendez, E., 1939.

Selenin, W. F., & Fogelson, L. I., Med. Biol. J. 1927. Ref. to by Fogelson, L. I., 1933.

Smith, J. R., Observations on the mechanism of the physiologic third heart sound. Am. Heart J. *28*: 661, 1944.

Steinberg, L. D., Über die normale dreigliederige Melodie des Herzens bei Kindern. Ztschr. f. Kinderh. *40*: 620, 1925.

Taquini, A. C., Les bruits cardiaques normaux par voie oesophagienne. Compt. rend. Soc. de biol. *125*: 534, 1937.

— & Braun-Menendez, E., Constance du bruit auriculaire par auscultation ou inscription oesophagienne. Ibid. *120*: 728, 1935.

Thayer, W. S., Further observations on the third heart sound. Arch. Int. Med. *4*: 297, 1909.

WARBURG, E., Om constrictio cordis. Ugeskr. f. læger *101*: 433, 1939. Ref. to by Frost, J., 1944.

WASSERMEYER, H., & ROHRBACH, A., Über chemische und kolloidchemische Prozesse im Herzmuskel bei experimenteller Myokarditis. Arch. f. exper. Path. u. Pharmakol. *166*: 375, 1932.

WEISS, S., Occidental beriberi with cardiovascular manifestations; its relation to thiamin deficiency. J. A. M. A. *115*: 832, 1940. Ref. to by Frost, J., 1940.

WHITE, P. D., The clinical significance of gallop rhythm. Arch. Int. Med. *41*: 1, 1928.

VIERORDT, O., Diagnostik der inneren Krankheiten. Leipzig, F. C. W. Vogel, 1888 (s. 182).

WIKNER, E., Hjärtats tredje ton, normal och patologisk galopprytm. Efter ett föredrag i Svenska Läkarsällskapet 30 sept. 1930. Hygiea *92*: 922, 1930.

WOLFERTH, C. C., & MARGOLIES, A., Gallop rhythm and the physiological third heart sound. Am. Heart J. *8*: 441, 1933.

— Extra heart sounds and frictions and their differential diagnosis. New Internat. Clin. *1*: 186, 1940.

WYSS, W. v., Aufzeichnung von Herztönen mit dem Einthoven'schen Saitengalvanometer und Untersuchungen über Galopprhythmus. Deutsches Arch. f. klin. Med. *101*: 1, 1910.

APPENDIX

Case history

Rabbit 1. ♀ 2 330 grammes. Injection 9. 12. 1943.

Time of examination	ECG.			PCG. Gallop rhythm
	T_1	T_2	T_3	
Before inj.	pos.	pos.	pos.	—
2 days after inj.	»	»	»	—
5 » » »	»	»	»	—
7 » » »	»	»	»	—
9 » » »	inverted	diphasic	»	—
12 » » »	pos.	pos.	isoel.	—
6 weeks » »	isoel.	»	pos.	—
8 » » »	pos.	»	»	—

Died 2. 2. 1944 after injection of acetylcholine. Observation period: 8 weeks. Microscopical examination: Slight, diffuse increase of connective tissue in the left ventricle.

Rabbit 2. ♀ 2 960 grammes. Injection 9. 12. 1943.

Time of examination	ECG.				PCG. Gallop rhythm
	T_1	T_2	T_3	Changes in S—T segments	
Before inj.	pos.	pos.	pos.	—	—
2 days after inj.	diphasic	»	»	—	—
4 » » »	»	diphasic	diphasic	—	—
7 » » »	pos.	pos.	pos.	+	—
9 » » »	»	»	isoel.	+	—
12 » » »	diphasic	diphasic	pos.	+	—
3 weeks » »	»	»	isoel.	+	—
6 » » »	pos.	pos.	»	—	—
8 » » »	»	»	»	—	—
16 » » »	»	»	»	—	—

Killed 1. 4. 1944. Observation period: 16 weeks.
Microscopical examination: Not performed.

Rabbit 3. ♀ 2 300 grammes. Injektion 15. 4. 1944.

Time of examination	ECG.			PCG. Gallop rhythm
	T_1	T_2	T_3	
Before inj.	pos.	pos.	isoel	—
6 days after inj.	inverted	»	pos.	—
9 » » »	diphasic	»	»	—
13 » » »	»	»	»	—
20 » » »	pos.	»	»	—

Killed 22. 5. 1945. Observation period 5 weeks.
Weight of animal 2 570 grammes. Weight of heart 7.0 grammes. Relative heart weight 2.72.[1]
Microscopical examination: Marked fibrosis and numerous confluent connective tissue scars of varying size in the left ventricular wall; no definite changes in the right auricle.

[1] Note: Multiplied by 1000 for convenience.

Rabbit 4. ♀ 3 350 grammes. Injection 18. 4. 1944.

Time of examination	ECG.			PCG. Gallop rhythm
	T_1	T_2	T_3	
Before inj.	pos.	pos.	isoel.	—
3 days after inj.	»	»	»	—
6 » » »	inverted	diphasic	»	—
10 » » »	»	»	»	—
15 » » »	»	pos.	»	—

Killed 22. 5. 1944. Observation period 5 weeks. Weight of animal 3 350 grammes. Weight of heart 10.0 grammes. Relative heart weight 3.0.
Microscopical examination: Fairly extensive, diffuse increase of connective tissue in one half of the left ventricle.

Rabbit 5. ♀ 2 800 grammes. Injection 18. 4. 1944.

Time of examination	ECG.			PCG. Gallop rhythm
	T_1	T_2	T_3	
Before inj.	pos.	pos.	isoel.	−
3 days after inj.	inverted	inverted	pos.	−
6 » » »	»	»	»	(+)
10 » » »	»	»	»	+

Died 28. 4. 1944 after injection of acetylcholine. Observation period 10 days.

Microscopical examination: Marked, diffuse increase of connective tissue in the left ventricle.

Rabbit 6. ♀ 3 250 grammes. Injection 18. 4. 1944.

Time of examination	ECG.			PCG. Gallop rhythm
	T_1	T_2	T_3	
Before inj.	pos.	pos.	inverted	−
3 days after inj.	»	»	isoel.	−
6 » » »	»	»	»	+
8 » » »	»	»	»	−
10 » » »	»	»	»	+
15 » » »	diphasic	»	pos.	+
17 » » »	pos.	»	isoel.	+
22 » » »	»	»	diphasic	−

Killed 22. 5. 1944. Observation period 5 weeks. Weight of animal 3 500 grammes. Weight of heart 10.0 grammes. Relative heart weight 2.86.

Microscopical examination: Slight diffuse increase of connective tissue in one or two larger areas of the left ventricle, in some places accumulating to minor fibrotic areas.

Rabbit 7. ♀ 4 300 grammes. Injection 18. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm	
					Frontal	Latera
Before inj.	pos.	pos.	pos.	–	33	39
3 days after inj.	»	»	inverted	+		
6 » » »	inverted	»	pos.	–		
10 » » »	isoel.	»	»	+		
11 » » »					32	41
15 » » »	isoel.	pos.	pos.	–		
21 » » »					37	39
22 » » »	pos.	pos.	pos.	–		
30 » » »	»	»	»	–		
31 » » »					36	36

Killed 21. 8. 1944. Observation period 5 weeks. Weight of animal 3 610 grammes. Weight of heart 12.3 grammes. Relative heart weight 3.04.

Microscopical examination: Pronounced, diffuse increase of a comparatively loose connective tissue, in places accumulating into fibrotic areas, mainly in the left ventricle but also in the right one. No hypertrophy.

Rabbit 8. ♀ 2 350 grammes. Injection 18. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	diphasic	–	29	32
3 days after inj.	»	»	inverted	–		
6 » » »	»	»	»	–		
10 » » »	»	»	»	–		
11 » » »					34	35
16 » » »	pos.	pos.	inverted	–		
22 » » »	»	»	pos.	–		
30 » » »	»	»	»	–		
31 » » »					32	37

Killed 21. 8. 1944. Observation period 5 weeks. Weight of animal 2 920 grammes. Weight of heart 9.2 grammes. Relative heart weight 3.15.

Microscopical examination: No definite pathological signs.

Rabbit 9. ♀ 3 050 grammes. Injection 18. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	diphasic	—	31	
5 days after inj.	»	»	»	—		
10 » » »	»	»	»	—		
11 » » »					31	32
16 » » »	pos.	pos.	diphasic	—		
22 » » »	»	»	»	—		
30 » » »	»	»	»	—		
31 » » »					32	32

Killed 21. 8. 1944. Observation period 5 weeks. Weight of animal 2 980 grammes.

Weight of heart 6.6 grammes. Relative heart weight 2.21.

Microscopical examination: But a very slight diffuse increase of connective tissue, or no definite pathological signs.

Rabbit 10. ♂ 2 650 grammes. Injection 18. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	isoel.	isoel.	—	28	29
3 days after inj.	»	pos.	»	—		
6 » » »	»	»	inverted	—		
10 » » »	»	»	»	—		
16 » » »	isoel.	»	isoel.	—		
17 » » »					27	30
22 » » »	pos.	pos.	pos.	—		
30 » » »	»	»	isoel.	—		
31 » » »					27	31

Killed 21. 8. 1944. Observation period 5 weeks. Weight of animal 2 640 grammes.

Weight of heart 7.5 grammes. Relative heart weight 2.08.

Microscopical examination: No definite pathological signs.

Rabbit 11. ♂ 2 700 grammes. Injection 18. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	neg.	—	33	31
3 days after inj.	»	»	pos.	—		
6 » » »	»	»	»	—		
10 » » »	inverted	isoel.	»	—		
15 » » »	»	pos.	»	—		
17 » » »					37	33
22 » » »	inverted	pos	pos.	—		
30 » » »	pos.	»	»	—		
31 » » »					36	33

Killed 21. 8. 1944. Observation period 5 weeks. Weight of animal 2 620 grammes.

Weight of heart 9.6 grammes. Relative heart weight 3.61.

Microscopical examination: In a few places a slight increase of connective tissue. Slight hypertrophy.

Rabbit 12. ♂ 2 900 grammes. Injection 28. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	inverted	—	29	29
5 days after inj.	inverted	inverted	pos.	+		
6 » » »	»	»	isoel.	+		
7 » » »					40	38
10 » » »	inverted	»	pos.	+		
13 » » »	»	»	»	+		
18 » » »	»	»	»	+		
24 » » »	»	»	»	+	40	38
29 » » »	»	»	»	+	39	34
33 » » »	»	»	»	+		

Killed 31. 8. 1944. Observation period 5 weeks. Weight of animal 2 680 grammes.

Weight of heart 9.1 grammes. Relative heart weight 3.04.

Microscopical examination: Fairly pronounced, diffuse increase of connective tissue in the left ventricle; none in the right ventricle. Considerable hypertrophy.

Rabbit 13. ♂ 2 600 grammes. Injection 28. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
					Size of cardiac shadow in mm.	
	T_1	T_2	T_3	Gallop rhythm	Frontal	Lateral
Before inj.	pos.	pos.	inverted	−	26	30
4 days after inj.	inverted	isoel.	pos.	+		
5 » » »	»	»	»	+		
6 » » »	»	»	»	+		
7 » » »					40	39
10 » » »	»	pos.	pos.	+		
13 » » »	»	isoel.	»	+		
18 » » »	»	»	»	+		
24 » » »	»	diphasic	»	+	42	40
29 » » »	»	»	»	+	38	35
31 » » »	»	»	»	+		

Killed 31. 8. 1944. Observation period 5 weeks. Weight of animal 2 440 grammes.

Weight of heart 9.2 grammes. Relative heart weight 3.07.

Microscopical examination: Fairly pronounced, diffuse increase of connective tissue in the left ventricle; practically none in the right ventricle. Considerable hypertrophy.

Rabbit 14. ♂ 2 950 grammes. Injection 28. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
					Size of cardiac shadow in mm.	
	T_1	T_2	T_3	Gallop rhythm	Frontal	Lateral
Before inj.	pos.	pos.	pos.	−	29	31
4 days after inj.	inverted	inverted	diphasic	+		
5 » » »	»	»	inverted	+		
7 » » »					41	42
8 » » »	isoel.	diphasic	diphasic	+		
10 » » »	»	»	»	+		
13 » » »	»	»	»	+		
18 » » »	diphasic	inverted	inverted	−		
24 » » »	»	»	»	+	46	42
29 » » »	pos.	diphasic	»	−	46	38

Killed 26. 8. 1944. Observation period 4 weeks. Weight of animal 2 740 grammes.

Weight of heart 10.7 grammes. Relative heart weight 3.09.

Microscopical examination: Fibrotic areas in two papillary muscles and diffuse increase of connective tissue in the right ventricle. Slight hypertrophy.

Rabbit 15. ♂ 2 250 grammes. Injection 28. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	isoel.	pos.	pos.	—	33	29
4 days after inj.	diphasic	diphasic	diphasic	—		
5 » » »	»	»	»	—		
8 » » »	»	inverted	inverted	—		
10 » » »	»	»	»	—		
11 » » »					40	35
13 » » »	diphasic	inverted	inverted	—		
18 » » »	isoel.	isoel.	isoel.	—		
24 » » »	»	»	»	—		
30 » » »	pos.	pos.	»	—		

Killed 31. 8. 1944. Observation period 5 weeks. Weight of animal 2 410 grammes.

Weight of heart 9.0 grammes. Relative heart weight 3.73.

Microscopical examination: Several considerable fibrotic areas in the right ventricle, only one or two in the left one. No hypertrophy.

Rabbit 16. ♂ 3 050 grammes. Injection 28. 7. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	inverted	—	33	31
4 days after inj.	»	»	diphasic	—		
8 » » »	»	»	»	—		
10 » » »	»	»	isoel.	—		
11 » » »					29	32
13 » » »	pos.	pos.	inverted	—		
18 » » »	»	»	isoel.	—		
24 » » »	»	»	inverted	—		
31 » » »	»	»	»	—		

Killed 31. 8. 1944. Observation period 5 weeks. Weight of animal 2 840 grammes.

Weight of heart 7.8 grammes. Relative heart weight 2.07.

Microscopical examination: Very slight diffuse increase of connective tissue in right and left ventricles. No hypertrophy.

Rabbit 17. ♂ 2 690 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	inverted	—	30	30
1 day after inj.	»	»	»	—	31	30
2 days » »	»	»	»	—	31	30
4 » » »	»	»	»	—	32	29
6 » » »	»	»	isoel.	—	31	29
9 » » »	»	»	inverted	—	31	30
11 » » »	»	»	isoel.	—		
16 » » »	»	»	pos.	—		31
6 weeks » »	»	»	»	—		

Killed 28. 10. 1944. Observation period 7 weeks. Weight of animal 3 120 grammes.

Weight of heart 9.1 grammes. Relative heart weight 2.91.

Microscopical examination: No definite pathological signs.

Rabbit 18. ♂ 2 490 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	pos.	—	31	34
1 day after inj.	diphasic	»	»	—	31	31
2 days » »	isoel.	»	»	—	32	31
4 » » »	»	»	»	—	34	31
6 » » »	inverted	»	»	—	39	37
9 » » »	»	»	»	—	41	36
11 » » »	isoel.	»	»	—		
16 » » »	inverted	»	»	—		32
6 weeks » »	pos.	»	»	—	34	31
7 » » »	inverted	isoel.	»	—		

Killed 3. 11. 1944. Observation period 8 weeks. Weight of animal 2 450 grammes.

Weight of heart 9.5 grammes. Relative heart weight 3.88.

Microscopical examination: Very slight increase of connective tissue in a minor area of the left ventricle. Considerable hypertrophy.

Rabbit 19. ♂ 2 700 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	inverted	—	34	31
1 day after inj.	inverted	isoel.	pos.	—	34	31
2 days » »	»	»	»	—	32	30
4 » » »	diphasic	diphasic	»	—	34	31
6 » » »	inverted	»	»	—	39	31
9 » » »	»	»	»	+	45	37
11 » » »	»	»	»	—		
16 » » »	isoel.	»	»	—	40	35
6 weeks » »	pos.	pos.	»	—	35	31

Killed 3. 11. 1944. Observation period 8 weeks. Weight of animal 2 850 grammes.

Weight of heart 9.1 grammes. Relative heart weight 3.19.

Microscopical examination: Several minor fibrotic areas in the left ventricle, none in the right one. Considerable hypertrophy.

Rabbit 20. ♂ 2 770 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	isoel.	—	36	34
1 day after inj.	»	»	»	—	34	30
2 days » »	»	»	»	—	33	31
4 » » »	»	»	»	—	36	31
6 » » »	»	»	»	—	34	33
9 » » »	»	»	»	—	37	34
11 » » »	»	»	»	—		
16 » » »	»	»	»	—	38	30
6 weeks » »	»	»	»	—		

Killed 28. 10. 1944. Observation period 7 weeks. Weight of animal 3 440 grammes.

Weight of heart 10.3 grammes. Relative heart weight 3.0.

Microscopical examination: No definite pathological signs.

Rabbit 21. ♂ 2 800 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	isoel.	pos.	pos.	—	31	31
1 day after inj.	diphasic	diphasic	»	—	31	31
2 days » »	»	»	»	—	31	31
4 » » »	inverted	»	»	—	31	30
6 » » »	»	»	»	+	38	33
9 » » »	»	»	»	—	35	36
16 » » »	»	»	»	+	41	36
6 weeks » »	»	inverted	»	+	31	30
7 » » »	»	»	»	—		

Killed 3. 11. 1944. Observation period 8 weeks. Weight of animal 2 950 grammes.

Weight of heart 8.4 grammes. Relative heart weight 2.85.

Microscopical examination: Slight diffuse increase of connective tissue in the left ventricle, none in the right one. Considerable hypertrophy.

Rabbit 22. ♂ 3 000 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	isoel.	—	31	32
1 day after inj.	isoel.	»	pos.	—	36	34
2 days » »	pos.	»	»	—	35	35
4 » » »	»	»	»	—	40	37
6 » » »	isoel.	»	»	—	46	38
9 » » »	inverted	diphasic	»	—	46	41

Died 21. 9. 1944. Observation period 11 days.

Microscopical examination: Marked diffuse increase of fresh connective tissue, mainly in the right ventricle but also in the adjacent parts of the left one. No hypertrophy.

Rabbit 23. ♂ 2 560 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	pos.	—	34	31
1 day after inj.	»	»	»	—	33	32
2 days » »	isoel.	»	»	—	33	32
4 » » »	diphasic	»	»	—	33	31
6 » » »	»	»	»	—	33	30
9 » » »	»	»	»	—	33	31
16 » » »	pos.	»	»	—	33	30
6 weeks » »	»	»	»	—		

Killed 28. 10. 1944. Observation period 7 weeks. Weight of animal 2 770 grammes.

Weight of heart 5.6 grammes. Relative heart weight 2.02.

Microscopical examination: No definite pathological signs.

Rabbit 24. ♂ 2 640 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	pos.	—	32	31
1 day after inj.	»	»	»	—	33	33
2 days » »	diphasic	»	»	—	33	31
4 » » »	inverted	»	»	—	32	31
6 » » »	»	»	»	—	39	31
9 » » »	»	»	»	—	39	35
16 » » »	»	»	»	—		34
6 weeks » »	»	»	»	—	34	34
7 » » »	»	»	»	—		

Killed 3. 11. 1944. Observation period 8 weeks. Weight of animal 2 860 grammes.

Weight of heart 9.2 grammes. Relative heart weight 3.22.

Microscopical examination: In the left ventricle, an insignificant increase of connective tissue in one or two areas. Considerable hypertophy.

Rabbits 25. ♂ 2 510 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	isoel.	—	32	31
1 day after inj.	»	»	»	—	29	30
2 days » »	»	»	»	—	31	31
4 » » »	»	»	»	—	30	32
6 » » »	»	»	»	—	31	29
9 » » »	»	»	»	—	32	31
16 » » »	»	»	pos.	—	30	31
6 weeks » »	»	»	»	—		

Killed 28. 10. 1944. Observation period 7 weeks. Weight of animal 2 810 grammes.

Weight of heart 9.2 Relative heart weight 3.31.

Microscopical examination: No definite pathological signs.

Rabbit 26. ♂ 2 240 grammes. Injection 10. 9. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	pos.	—	31	29
1 day after inj.	inverted	»	»	—	31	29
2 days » »	»	diphasic	»	—	33	28
4 » » »	»	»	»	—	33	29
6 » » »	»	inverted	»	—	34	30
9 » » »	»	»	»	—	34	30
16 » » »	»	»	»	—	34	31
6 weeks » »	»	pos.	»	—		

Killed 28. 10. 1944. Observation period 7 weeks. Weight of animal 2 390 grammes.

Weight of heart 7.5 grammes. Relative heart weight 3.14.

Microscopical examination: In the left ventricle small areas of diffusely increased connective tissue, none in the right one. Considerable hypertrophy.

Rabbit 27. ♂ 2 400 grammes. Injection 11. 11. 1944.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	pos.	−	30	25
6 days after inj.	inverted	»	»	+	42	34
7 » » »	»	»	»	+	42	
8 » » »	pos.	»	»	+		
9 » » »	»	»	»	+		
10 » » »	»	»	»	+		
11 » » »	»	»	»	+	38	33
12 » » »	»	»	»	+		
15 » » »	»	»	»	+	39	33

Killed 25. 11. 1944. Observation period 15 days. Weight of animal 2 450 grammes.

Weight of heart 8.1 grammes. Relative heart weight 3.31.

Microscopical examination: Slight diffuse increase of connective tissue in the left ventricle; extensive formation of fibrotic areas in the right ventricle. No hypertrophy.

Rabbit 28. ♂ 2 200 grammes. Injection 4. 2. 1945.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	isoel.	−	26	31
4 days after inj.	inverted	isoel.	pos.	−	46	38
5 » » »	»	»	»	+		
11 » » »	»	pos.	»	+	42	35
16 » » »	»	»	»	−	39	38
3 weeks » »				−	37	38
5 » » »	inverted	pos.	pos.	−	38	35
7 » » »	»	»	»	−	39	34

Killed 28. 3. 1945. Observation period 7 weeks. Weight of animal 2 760 grammes.

Weight of heart 9.7 grammes. Relative heart weight 3.51.

Microscopical examination: A few considerable fibrotic areas in the left ventricle; slight diffuse increase of connective tissue in the right ventricle. Considerable hypertrohpy.

Rabbit 29. ♀ 2 550 grammes. Injection 4. 2. 1945.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	inverted	—	28	32
4 days after inj.	inverted	isoel.	pos.	+	45	42
5 » » »	»	inverted	»	+		
11 » » »	»	diphasic	»	(+)	45	43
16 » » »	isoel.	pos.	»	—	44	41
3 weeks » »	pos.	»	»	—	38	36
5 » » »	»	»	»	—	33	37
7 » » »	»	»	»	—	30	33

Killed 28. 3. 1945. Observation period 7 weeks. Weight of animal 2 170 grammes.

Weight of heart 7.0 grammes. Relative heart weight 3.23.

Microscopical examination: One large and several smaller fibrotic areas in the left ventricle, slight diffuse increase of connective tissue in the right ventricle. Considerable hypertrophy.

Rabbit 30. ♀ 2 100 grammes. Injection 4. 2. 1945.

Time of examination	ECG.			PCG.	Röntgen	
	T_1	T_2	T_3	Gallop rhythm	Size of cardiac shadow in mm.	
					Frontal	Lateral
Before inj.	pos.	pos.	pos.	—	28	28
4 days after inj.	inverted	inverted	»	+	35	33
5 » » »	»	pos.	»	+		
11 » » »	diphasic	»	»	+	37	35
16 » » »	inverted	»	»	—	35	33
3 weeks » »	pos.	»	»	—	32	32
5 » » »	diphasic	»	»	—	31	32
7 » » »	» (inverted)	»	»	—	31	31

Killed 28. 3. 1945. Observation period 7 weeks. Weight of animal 2 180 grammes.

Weight of heart 6.7 grammes. Relative heart weight 3.07.

Microscopical examination: Two fairly big fibrotic areas in the left ventricle; slight diffuse increase of connective tissue in the right ventricle. Considerable hypertrophy.

From the Medical Department of the Children's Hospital, Gothenburg, and the Pediatric Clinic of Karolinska Institutet at Norrtull's Hospital, Stockholm, Sweden

RHEUMATIC FEVER WITH CHOREA MINOR

A CLINICAL STUDY WITH SPECIAL REFERENCE TO THE PROGNOSIS

BY

ERIK JACOBSSON

ACTA PÆDIATRICA, VOL. XXXIII, SUPPLEMENTUM VII

Stockholm 1946

Translated by Grace Lundberg, B. A.

UPPSALA 1946
ALMQVIST & WIKSELLS BOKTRYCKERI AB
40772

To my Mother and
the Memory of my Father

Preface.

My interest in rheumatic fever was first aroused in 1942, when, in connection with my work as assistant physician to Prof. A. Wallgren at the Children's Hospital, Gothenburg, I began to realize how prevalent this disease was among children, and not least among the child population of Gothenburg. I therefore decided to make a closer study of the subject, and by assembling the cases treated at the hospital and submitting them to a follow-up examination to offer my contribution towards clearing up a number of the problems connected with this disease, especially as regards its prognosis.

To my chief, Professor Arvid Wallgren, who followed my investigations with never-failing interest, and during the course of my work was always ready to offer stimulating advice and encouragement, I owe a great debt of gratitude.

The studies presented in this book were carried out during the years 1943—1946 at the Medical Department of the Children's Hospital, Gothenburg, and at the Pediatric Clinic of Karolinska Institutet at Norrtull's Hospital, Stockholm.

The roentgen examinations of the heart were carried out by Dr. C. J. Hansson, chief roentgenologist of the Children's Hospital, Gothenburg. I am extremely grateful to him for his interest and valuable assistance, as well as for all the trouble he went to on my account.

To Docent L. Goldberg, who made many useful suggestions and gave me invaluable help in connection with the statistical side of my studies I proffer my grateful thanks.

Fil. lic. B. Matérn very kindly assisted me in planning and making the calculations in the chapter on the recurrence rate. His wide knowledge in the fields of insurance and mathematics were placed unreservedly at my disposal, and I wish to take this

opportunity of expressing my appreciation of his helpful contribution.

To Med. Dr. Gert von Sydow, acting chief at the Children's Hospital, Gothenburg during part of the period during which my follow-up investigation was in progress, and to Med. Dr. Yngve Åkerrén, the present acting chief of the hospital, I am indebted for pleasant working conditions during the course of my investigations as well as for the friendly advice and kindly interest they took in my work.

For valuable discussions and suggestions I wish to thank my colleagues at the Children's Hospital in Gothenburg and at Norrtull's Hospital in Stockholm. I especially desire to express my thanks to Dr. John Lind, with whom I discussed the electrocardiographic tracings and alterations.

In conjunction with their daily routine work at the hospital, a number of nurses from the laboratory and roentgen departments of the Gothenburg Children's Hospital were kind enough to help me by carrying out the vast majority of the laboratory tests and roentgen examinations. I should especially like to mention here Mrs. Sonja Jönsson, Miss Dagny Hedberg, and Miss Elisabeth Wallberg.

I was also greatly assisted by Mrs. Mia Dahlén, who did most of the checking of the hospital case records and of the deskwork, and persuaded many of the patients to submit themselves for examination; for all this I am much obliged to her.

The investigation was carried out with the aid of grants from "Stiftelsen Therese och Johan Anderssons Minne" and "Konung Gustaf V:s 80-årsfond".

Finally, I wish to thank my wife for all the help she has given me in so many ways in connection with this work. Her untiring support and interest have acted as a constant stimulation to my efforts.

Stockholm, December 1946.

ERIK JACOBSSON.

Introduction.

During the course of the centuries, rheumatic fever has been described under a variety of different names [rheuma or katarrhous (Hippocrates), rheumatismus (Ballonius), polyarthritis acuta, polyarthritis subacuta, polyarthritis subchronica, polyarthritis rheumatica acuta, rheumatismus infectiosus specificus, morbus rheumaticus specificus, rheumatismus verus, acute arthritis, rheumatoid arthritis, acute rheumatic arthritis, rheumatism, rheumatic fever, Bouillaud's disease, acute articular rheumatism, and corresponding synonyms in various languages]. Chorea minor with its various names — St. Vitus' dance, Sydenham's chorea, chorea rheumatica, etc. — has been included by some authors as a major manifestation of rheumatic fever, and by others not included at all. All these designations stressed to a greater or lesser extent the significance of various symptoms, or reflected the current view as to the dominant feature of the disease. A number of different diseases were originally concealed in the syndromes indicated by these names; as time went on, however, many of them were separated from rheumatic fever, which thus gradually emerged as a fairly typical disease with a definite and characteristic complex of symptoms, readily distinguishable, as a rule, from the others.

According to Chomel (1837), Hippocrates himself gave a description of acute arthritis, but in his conception, rheuma or katarrhous, not only all types of joint disease were included but also various diseases in the viscera and extremities. It was not until the seventeenth century, when Guillaume de Baillou or Ballonius attached a purely symptomatic significance to the name and included in it all diseases with flowing pains in the peripheral parts of the body, particularly the joints and muscles, that the term rheumatism was used to describe acute polyarthritis as a

separate disease (cited from Wilson, 1940). When Sydenham in 1676 distinguished gout as a separate disease, a more satisfactory and in many respects excellent clinical description of acute polyarthritis was obtained. A few years later, Sydenham published his report on chorea minor but it was not till 1809 that Bouteille, in a monograph on chorea, described one case of his own and four assembled from the literature in which chorea occurred in association with rheumatic arthritis (in four of the patients the condition developed immediately after the disappearance of the arthritic symptoms while in the fifth chorea preceded the joint affection). Although Begbie, in 1847, on the basis of his own clinical experience and that of his colleagues demonstrated that the three manifestations, arthritis, chorea, and carditis can "occur conjointly or severally in different individuals in the same family", it was chiefly thanks to Sée, in 1850, and Roger, in 1867, that the intimate connection between chorea and rheumatic fever was established. Thus, Roger maintained that chorea, polyarthritis, and endocarditis must be regarded as three different names for one and the same disease.

The connection between rheumatic fever and heart disease was pointed out for the first time by Pitcairn in 1778, who stated that "persons the subject of rheumatism were attacked more frequently than others with symptoms of organic disease of the heart". The more intimate connection between rheumatism and heart disease did not, however, become more generally known, until after Bouillaud had stated his famous Law of Coincidence: "In the great majority of cases of diffuse acute articular rheumatism with fever, there exists in a variable degree a rheumatism of the sero-fibrous tissue of the heart. The coincidence is the rule, and the non-coincidence the exception" (Bouillaud, 1840).

The tendency of rheumatic fever to occur predominantly in childhood was mentioned for the first time by Sir Thomas Watson (1835), who also stated that the younger the patient the more likely it is that rheumatic heart disease will develop, the risk becoming less, however, after adolescence, as the age increases. In the early days, endocarditis and pericarditis were the only conditions to which attention was paid, but in the second half of

the nineteenth century inflammatory lesions in the myocardium were observed, their specificity (the Aschoff bodies) being established later by Aschoff in 1904.

According to Hedley (1940), John Haygarth was the first to write a monograph (1888) on rheumatic fever, and he also was the first investigator who used the name rheumatic fever for this disease.

The first time rheumatic subcutaneous nodules were mentioned in the literature was in 1812, by Wells; Hillier, in 1868, was the first to describe nodules in children. The close connection between these nodules, and rheumatic fever and chorea, was shown in 1881, by Barlow and Warner, and a few years later it was still further stressed by Cheadles (cited from Wilson, 1940) in his description in 1888 of "The Various Manifestations of the Rheumatic State as Exemplified in Childhood and Early Life".

The first description of what is now (after Leiner) called erythema annulare was furnished in 1831 by Bright, who had observed cases of "roseola annulata" in association with chorea.

Thus, although the disease has been known for a long time, it was not till the last half-century that its predilection for the early years of life was fully realized. This would seem in part to be the reason why the prognosis, particularly the late prognosis for those contracting rheumatic fever during childhood, has been the subject of so few investigations. Added to this, we have the fact that it is only during more recent years that there have been consistent attempts to distinguish between primarily chronic polyarthritis (including also its primarily acute form) and rheumatic fever, this being the reason why a well-defined series of patients with rheumatic fever, under observation for a satisfactorily long period, has only lately become available as a subject of study from the standpoint of the prognosis.

Criteria for diagnosis.

Although many attempts have been made to find a reliable diagnostic aid, no one has yet succeeded in working out a method capable of proving whether rheumatic fever has previously been in existence or is at the moment active, a circumstance which must in part be ascribed to our deficient knowledge as to the true provoking factor in the disease. In making the diagnosis it therefore has been found necessary to rely on various signs and symptoms or combinations of these, and in consequence, absolute unanimity as regards diagnosis has not been reached among the various authors. The result of this has been that some investigators have made the rules for diagnosis too narrow, others, particularly earlier authors, have made them too wide. Owing to this, difficulties arise when an attempt is being made to compare the various investigations with one another.

In view of what has been said above, it is necessary to define clearly the criteria required to-day for making a diagnosis of rheumatic fever. These criteria on the whole, are in close agreement with those recently published by Duckett Jones (1944). It should at once be clearly stated that, as a rule, no single manifestation alone can be regarded as conclusive evidence of the presence of rheumatic fever; it is only when two or more manifestations are present in combination that the probability of a proper diagnosis is increased to a greater or lesser degree. Certain manifestations must, naturally, be attributed greater significance than others, and of these, which are called major manifestations by American investigators, only a combination of two is necessary as a rule, particularly as several of the minor manifestations of rheumatic fever are usually present at the same time and thus contribute towards making a diagnosis of rheumatic fever reasonably certain. On the other hand, if only one of the major mani-

festations is found, then the number of the minor signs and symptoms must be proportionately increased, or else those present must be of a type considered to have important diagnostic significance. The presence of minor manifestations alone is not sufficient to justify a diagnosis of rheumatic fever, although a combination of several of the more significant ones is enough to cause rheumatic fever to be suspected. It should be stressed, however, that these criteria need by no means be considered unchangeable for all time, at least as far as the minor manifestations are concerned; the diagnostic significance of the latter will in all probability undergo some changes in proportion as our knowledge of this disease increases.

I. The *major manifestations* are arthritic symptoms, including arthralgia; carditis; chorea; subcutaneous nodules; erythema annulare; and a verified statement as to a previous attack of rheumatic fever or a later attack of the same disease with undeniable signs and symptoms.

II. The *minor manifestations* are usually considered to be as follows: fever; increase in the sedimentation rate of the red blood cells; anemia; increase in the number of the white blood cells; and in addition a number of other fairly irregular signs or symptoms.

I. Comments on the major manifestations.

As regards *arthritic symptoms*, these are too well known to need discussion. It should be remembered, however, that signs and symptoms from the joints are often very mild in children, a fact which has been pointed out by a number of authors (Cheadle, 1889; Findlay, 1931; Vaubel, 1938; Fanconi and Wissler, 1938; May Wilson, 1940, and others). Typical features of these arthritic lesions in children are their evanescent nature, their tendency to flow from joint to joint, and the fact that in many instances there are only subjective symptoms, and no definite objective signs.

It is of importance in the evaluation of the arthritis not to forget to rule out other acute or chronic causes of joint affection in children such as various forms of infectional arthritis, scarlatina,

tuberculosis, syphilis, gonorrhea, serum sickness, osteomyelitis, leukemia, Still's disease, primary chronic polyarthritis, and so on. Thus, patients with signs and symptoms suggestive of chronic polyarthritis right from the beginning of the disease should not be regarded as cases of rheumatic fever — a rule which needs to be stressed, as it is still sinned against in some quarters.

The signs from *the heart* in the early stages of rheumatic fever can be said to consist, as a rule, of *valvulitis* (endocarditis) and *myocarditis*. The former condition announces its presence by murmurs which change character often from day to day or from hour to hour, indicating the existence of an active process in the valves (or naturally of myocarditis of greater or lesser extent with the resulting more or less marked and varying dilatation of one or more of the heart chambers). It is only after the endocarditic process has progressed, and when more stationary lesions in the valves, valvular deformities in the form of adhesions, shrinking of the cusps, or fibrotic proliferation, have appeared, that the murmur caused by the valvular affection can be expected to become more constant. According to Coombs, we must count on the mitral valves being affected in all cases of rheumatic carditis, the aortic valves in half the cases, and the tricuspid valves in one-third, while the pulmonary valves are rarely attacked. (Of 97 cases submitted to postmortem examination, affection of the mitral valve was observed by him in all of them, the aorta was involved in 57, the tricuspid valve in 35 and the pulmonary valve in 2 cases.) Cabot (1926) found that among 208 cases of rheumatic valvular heart disease the mitral valve was involved in 178 cases, the aorta in 93 cases, the tricuspid in 33, and the pulmonary valve in 4.

Rheumatic myocarditis in its milder forms is often difficult or impossible to detect, whereas the more advanced forms are clearly distinguishable clinically through such signs as dilatation, signs of failing cardiac function with enlargement of the liver, dyspnea, cyanosis, edema, tachycardia and so on. Aside from the electrocardiographic changes, the milder forms seem to manifest themselves only by a moderate degree of tachycardia, extra-systoles, impure or weakened heart tones, and evanescent systolic mur-

murs, signs which cannot be regarded as significant. When once the rheumatic process has begun to attack the conducting system we are afforded a better opportunity for objectively establishing the presence of myocarditis by means of the electrocardiogram. The commonest form of electrocardiographic lesion then observed is a prolongation of the auricular-ventricular conduction time, which may be more or less pronounced and which in the severe cases leads to a complete heart block. Other changes in the electrocardiographic tracings, such as abnormality of the QRS-complex, lowering or elevation of the S—T-segment, lowering, flattening, or inversion of the T-wave and so on, may also constitute evidence of myocardial implication. In a survey of the more recent American literature on these aspects Orgain and Martin (1941) established that electrocardiographic abnormalities were present in 40—100 per cent of children with rheumatic heart disease. Scherf and Boyd write as follows: "In rheumatic fever, electrocardiographic examinations, when repeated daily for awhile, have also shown that the myocardium is affected, at least temporarily, in the course of every rheumatic disease".

Although *pericarditis* may, in isolated cases, constitute the first evidence of rheumatic fever, this very rarely happens. On the other hand, it is by no means an uncommon occurrence later on in the course of the disease. According to Vaubel (1938), Coombs found pericarditis in 50 per cent of the patients in his adult autopsy material, although the clinical examination had only disclosed pericarditis in 13 per cent. In an autopsy material of 62 children with rheumatic pericarditis only 35 had shown clinical signs of pericarditis (Gibson et al., 1936), while May Wilson (1940), in her series of 26 children who had died of rheumatic fever, found 20 cases of pericarditis, 9 of which were among the 12 children dying at the initial attack (6 had been detected at the clinical examination) and 11 among the 14 who died during a recurrent attack (10 of these had been diagnosed on clinical grounds).

When pericarditis develops during the course of rheumatic fever the symptoms and physical signs of this affection are often the dominating feature of the clinical findings. On the other hand,

the signs of pericarditis, when they are present, can be so evanescent that the diagnosis cannot always be made in the living subject. The statements regarding the presence of pericarditis vary, therefore, with the different authors, the incidence mentioned ranging up to 31 per cent (Warburg, 1931).

The connection between rheumatic fever and chorea minor has, as already mentioned, been known for many years, and has been the subject of much discussion particularly as regards the correctness of regarding chorea as a manifestation of rheumatic fever. Many investigators would seem to be agreed that this is the correct interpretation (Osler, 1894; Coombs, 1924; Swift, 1931; Bland and Jones, 1935; Helmreich, 1935; Wilson, 1940, and others). A few of the more recent authors, however, are unwilling to admit that chorea minor is a form of rheumatic fever (Gerstley et al., 1935; Coburn et al., 1937). These authors do not, as a rule, deny that chorea occurs in association with, and as a result of, a rheumatic infection, but they also consider that the ground for chorea is prepared even without rheumatic fever, through the existence of other factors such as (Gerstley) psychic trauma, age, temperament, constitution, or possibly disorder of the glands of internal secretion.

The question of the more or less intimate relationship between chorea and rheumatic fever will probably continue to give rise to discussion and research for some time to come. "The answer to this difficult problem must," according to Jones and Bland (1935), "remain indefinite until more knowledge concerning the etiology is available". Although chorea minor may appear without any other manifestations of rheumatic fever, either in the previous history or in the subsequent course, it must nevertheless be regarded as fully justified for the present — so long as the circumstances of the case do not point undeniably in another direction (chorea gravidarum, Huntington's chorea, etc.) — to look upon chorea as a special form of rheumatic fever; a certain reservation should be made, however, with regard to the cases which neither present sure signs of carditis in connection with the attack of chorea nor have recurrent attacks of arthritis or rheumatic carditis, for instance, at a later date.

Although the diagnosis rheumatic fever must sometimes be regarded as doubtful, there seems good reason to expect that the subsequent course will provide the correct answer. For it is just its marked *tendency to recurrence* that has always been considered to be one of the most characteristic features of the disease. A recurrent attack of the same signs or symptoms or of others perhaps even more typical than the initial lesion, must be regarded as strong confirmatory evidence as to the correctness of a previous, more or less tentative, diagnosis (Wallgren, 1939; Duckett Jones, 1944, and others). The recurrence rate varies, naturally enough, in different reports, but on an average it may be said to be 70 per cent. [Roth, Lingg, and Whittemore noted at least one recurrence in 68 per cent (in a total of 488 cases) — in the chorea cases the rate was as high as 74 per cent; Kaiser stated in connection with a report on 1 200 cases of juvenile rheumatic fever that the rate of recurrence was more than 50 per cent; Duckett Jones mentioned a rate of about 70 per cent; Wilson observed one or more recurrent attacks in 85 per cent of a group of 673 children.] The variations in the different reports can probably be ascribed mainly to differences in the length of the observation period or to different criteria for judging a recurrence.

Subcutaneous nodules are stated by all investigators to be a very typical feature of rheumatic fever; many authors regard them as pathognomonic of this disease (Klinge, 1933; Chiari, 1938; Wallgren, 1939; Fanconi and Wissler, 1943; Bergstrand, 1943, and others) while others deny that there is any specificity (Findlay, 1931; Klinge, 1933, and others), pointing out that nodules occur also in cases of chronic polyarthritis (according to Dawson, in 25 per cent of 245 cases of rheumatoid arthritis), a statement which does not seem to have been proved beyond question, however. Their occurrence is very varying; they seem to be commonest in England and Eire (Wallgren, 1939) and in at least some parts of America (Wilson, 1940), and less usual in Germany (Fanconi and Wissler) and Sweden (Wallgren). The fact that they are often of small size and sometimes transient in nature causes them to be readily overlooked, and also perhaps affords an explanation of the disparity between the incidence figures reported in

various papers — according to a survey worked up by Dawson the percentage incidence ranges between 10 and 25—50 in cases with rheumatic fever, but lower figures have also been mentioned (7.5 per cent, according to Fanconi and Wissler). In Britain, the incidence is 25—50 per cent in London, while in a material from Glasgow, nodules were only present in about 10 per cent (Findlay, 1931). Not only do they show geographical variations as regards their occurrence but they may also vary from time to time (Findlay).

Erythema annulare (Leiner) is regarded by most authors as being a specific allergic manifestation of rheumatic fever. According to Lehndorff and Leiner, this sign must be looked upon as absolutely pathognomonic of the disease, and other authors also express the same opinion (Bindschedler, 1936; Debré et al., 1938; Wallgren, 1939; Fanconi and Wissler, 1943). This sign sometimes appears at a very early stage in a rheumatic infection, sometimes even before any other signs or symptoms have developed, and it need not necessarily be associated with other signs of the disease. It may often occur several weeks before other signs of a recurrent attack (Swift, cited from Vaubel). Unfortunately, however, it is a symptom very rarely observed, a circumstance to be explained chiefly by its highly evanescent nature, and only repeated inspection of the skin will lead to its detection in the individual cases (Wallgren). The erythema disappears in many cases shortly after it has been observed, but as a rule it appears again in the patient in question after an interval of varying length and may persist for several days. Its extremely evanescent nature and often modest appearance is probably the reason why, in many publications, it is reported as occurring in so few cases, if at all. Possibly, various geographical factors play a rôle in the case of this erythema as in that of the subcutaneous nodule. Investigators who have paid special attention to this sign give figures for the percentage incidence ranging between 3 and 15 (Fanconi and Wissler), but a figure as high as 62.5 per cent has been mentioned (Leichtentritt, 1930) while others who have not been particularly interested in it report only isolated cases, or else do not mention the sign as present at all in their cases (Find-

lay, 1931; Wilson, 1940; Friedländer, 1945). Many authors are of the opinion that the sign is an indication of incipient or established carditis, often of a severe nature (Leiner and Lehndorff, 1922; Leichtentritt, 1930; Friedländer, 1945), while in opposition to this view Wallgren does not consider the prognosis to be so bad when this exanthem is present, even though it usually occurs in connection with the severer forms of rheumatic fever. Fanconi and Wissler also found this sign "mehrmals als Begleitsymtom leichter Herzkrankheiten", but they admit that it is often a characteristic of the more advanced cases and that the combined occurrence of erythema annulare and rheumatic nodules is a particularly grave finding. Thus, Lehndorff and Leiner state that when this sign was present there was always an undoubted endocarditis or a murmur suggestive of a valvular affection; it occurs as a rule unaccompanied by other signs or symptoms and only in some cases is its occurrence a sign of a complication.

II. Comments on the minor manifestations.

The minor manifestations are signs or symptoms which are not specific to rheumatic fever to the same extent as the major manifestations; they may often occur also in association with other infections or in other combinations. One of these manifestations is *fever*, which, as a rule, is very pronounced but need not necessarily be so, at least not to any considerable degree. This is especially the case in chorea, in which fever is hardly ever present during the course. Jones and Bland (1935), for instance, interpret a fever occurring without any other apparent cause during an attack of chorea as a sign of some other manifestation of rheumatic fever.

Another rather significant sign is *the increase in the sedimentation rate of the red blood cells*, which is nearly always to be observed in rheumatic fever. This feature is, as a rule, very pronounced and of long duration, and the infection can hardly be said to be over before the sedimentation reaction is steadily normal again (Kahlmeter, 1928; Elghammar, 1934; Helmreich,

1935; Payne and Schlesinger, 1935). Strangely enough, the sedimentation rate is often not increased at all in chorea, a fact which has been considered to be evidence that chorea may not be a manifestation of rheumatic fever. The reason for the normal sedimentation rate in such cases is not clear.

The erythrocyte sedimentation rate has during the past ten years been used to an increasing extent as a sensitive and reliable measure of the activity of the rheumatic infection.

According to Wallgren, there are three situations in which the reaction in question cannot be used as a guide to the activity of the disease. In addition to chorea, this is the case in cardiac incompensation, when the sedimentation rate often decreases, and in upper respiratory tract infections in a rheumatic patient, when a previously fairly low sedimentation rate may increase and subsequently remain high for a long period after the disappearance of the acute infection.

An *increase in the number of leukocytes* in the blood is observed fairly often, although not always, in rheumatic fever. This abnormality disappears much sooner than the increased sedimentation rate. A raised leukocyte count appears to be less common in chorea (Fanconi and Wissler, 1943); at any rate, it is not so marked in chorea as it is in other forms of rheumatic fever (Wilson, 1940).

Another blood change not infrequently noted in rheumatic fever is a greater or lesser degree of *anemia*, with lowered hemoglobin values and a reduction in the number of red blood cells. If an increased sedimentation rate persists after acute infection of the upper respiratory tract during a rheumatic fever, the presence or absence of anemia may often be evidence of whether endocarditis or some other form of rheumatic activity is present or not (Wallgren). As a rule, this anemia is not particularly severe. Kahlmeter found anemia with an erythrocyte count of less than 4 million in 65 per cent of his cases, this value being obtained within a week in half of the cases; similar conditions were observed by Gezelius (1939). The anemia returns to normal sooner than the sedimentation rate.

Besides the above-mentioned signs, a great many others may

be present in a few cases. *Epistaxis* is fairly common; it may occur time after time in one and the same patient, and it then usually occurs in connection with exacerbations of the disease. Another striking feature often observed is the *pallor* of the patients. *A strong tendency to perspire*, *anorexia*, and *loss of weight*, are other symptoms often observed during the first, as well as in the severe, stages. Some patients have *pleurisy*, which, as a rule, is left-sided (secondary to the pericarditis?), others have *atelectatic* or *pneumonic changes in the lungs*, particularly in the more severe forms of the disease. In the earlier stages, *positive reactions for albumin in the urine* are found in some cases for a few days, and there is simultaneously present in about half the patients in the beginning stages of the disease (Nathhorst, 1940) an *erythrocyturia* which, as a rule, is never particularly severe, however, and never lasts longer than 1 or 2, or at the most 4, weeks.

In the acute stage of the disease a few patients complain of *abdominal pain*, which in some may be of such severity that appendicitis is suspected and a surgical procedure performed. In many of these patients it must be a question of pains originating from a hip joint affection, but in many others nothing of this kind can be demonstrated objectively. Abdominal pain arising during the course of the disease may, of course, be an expression of incipient or progressive pericarditis, with the pain situated down towards the abdomen. *Pain over the heart region* is present in some cases, although this symptom is, in general, not an uncommon one in childhood and early life, especially as a reflection of certain neurasthenic characteristics. *Pain in the back and nuchal region* may, in some cases, cause meningitis to be suspected, particularly if the patient has a stiff neck; in these cases the stiffness is apparently due, as a rule, to arthritis in the joints of the cervical vertebrae. Because of this pain, and particularly if there is also arthritis of the large joints of the legs, the children sometimes cannot move their legs and are therefore suspected to have poliomyelitis and sent to a hospital for epidemic diseases.

Because the general nature of the disease is one of mesenchymal affection, symptoms may arise in practically any organ or system

of organs in the body, often before the more obvious signs and symptoms of rheumatic fever have appeared. Thus, *skin lesions*, for instance, in the form of erythema nodosum, have been present without the patient in question being tuberculin-positive, and it has therefore been concluded that the exanthem in these cases was of rheumatic origin (Wallgren); it is, however, without doubt a commoner occurrence to find that a rheumatic person with erythema nodosum is also tuberculin-positive, although the tuberculous process need not necessarily be active.

Earlier investigations.

Age and sex incidence.

When studying the literature on rheumatic fever from the close of last century and the beginning of the present one, one cannot help being struck by the fact that the age at the onset of the disease in the majority of cases is stated to be over the actual age of childhood. Riess (1898), for instance, says that acute articular rheumatism occurs most frequently between the ages of 15 and 30, after which, patients between the ages of 30 and 50 are those most frequently attacked, while the incidence of the disease is relatively low between the ages of 5 and 15 years. According to Bäumler (1901), the incidence of the first attack is highest among persons between the ages of 15 and 25 years, while in very young children articular rheumatism certainly can occur, but is rare. In an even earlier investigation (Lebert, 1860), it is stated that 55 per cent (among 230 cases) occur between the ages of 16 and 25 years, 30 per cent between 26 and 40 years of age, and only 5 per cent in the years prior to adolescence. Similar figures are mentioned by Senator (1879), who also pointed out that articular rheumatism hardly ever occurs in children under five years old and that a first attack is extremely rare in persons over 50 years of age.

This fact, that true articular rheumatism was previously not considered to be one of the diseases predominantly occurring in the early years of life, can in all probability be put down mainly to the evanescent nature of the symptoms, particularly those from the joints, a fact which many of the later investigators have established. Furthermore, as there seems to have been little interest in children and childhood diseases in earlier centuries, it is not unlikely that a not inconsiderable number of cases of

rheumatic fever in children were overlooked or interpreted as some entirely different disease. That the figures mentioned, as compared with those obtained in the more recent investigations, might indicate a displacement in the age of predilection for rheumatic fever in the direction of younger ages is hardly probable. It should be remembered, also, that the signs and symptoms, as the result of all the recent investigations, are now more clearly distinguishable, and it is furthermore being realized more clearly that the subjective symptoms, especially those from the joints, are often extremely slight even in undeniable cases of rheumatic fever. There are, however, authors from earlier years, who considered that rheumatic fever occurs fairly frequently in children. Roger (1867), for instance, wrote as follows: "Le rhumatisme est une affection assez fréquente chez les jeunes sujets, contrairement à l'opinion généralement reçue".

On the other hand, there seems to be little difficulty in explaining why chorea, with its strikingly pronounced symptoms, aroused the interest of laymen and medical men alike even in previous centuries, not least as regards its occurrence in children. Thus Wollenberg, a contemporary of Bäumler and Riess, stated as early as in 1899, in a survey over a number of different papers, that the incidence of chorea is highest between the ages of 7 and 13 years.

Investigations of the past few decades, however, are demonstrating in ever-increasing numbers that rheumatic fever is a disease which is especially predominant in the years of childhood and adolescence, and that, as a rule, it is just during these years that the disease first appears. On the basis of a study of 172 cases which, however, includes only children under 12 years of age, Poynton, Paterson, and Spence (1920), for instance, stated that the peak age of incidence for the initial attack is 7 years, both as regards rheumatic fever and chorea. Findlay (1931) found in a group of 364 children with rheumatic arthritis in Glasgow, that in 52.2 per cent the disease made its first appearance between the ages of 7 and 10 years, and in 28.5 per cent between 11 and 14 years of age, whereas none of his patients had had the first attack before the third year of life. The same could on the

whole be said of chorea. Seven years represented the most common age for the first evidence of the disease both as regards rheumatic arthritis and chorea.

Among 343 cases of chorea minor, Nordgren (1922) observed that during the years 1908 to 1921 in Stockholm, Sweden, the frequency of the disease was at its highest between the ages of 6 and 12, with the peak at 8 to 10 years. He found 5 cases of 3—4 years old but none below this age (lowest age 3 years and 4 months).

Kaiser (1934), in an investigation on 1 126 cases of juvenile rheumatic fever in Rochester, found 33 cases under 2 years of age. From this age there was a gradual rise in frequency up to the tenth year, after which the incidence declined rapidly until, at the fifteenth year, it was as infrequent as in the early years of childhood. The figures mentioned by Fanconi and Wissler (1943) are on the whole the same, although, according to them, only a few cases are found under the age of 3. The peak incidence for chorea cases was stated to be at 11 years of age.

Shapiro (1936), in his material consisting of 713 children from Minnesota during the years 1932 to 1936, found that the curve showing the age at onset rose steeply to the peak at 5—6 years of age, a fact which he felt, after comparing the statements in older papers, should be ascribed to earlier hospital treatment, earlier diagnosis, and other such factors resulting from the present, more wide-spread knowledge of the disease.

Roth et al. (1937) maintained on the basis of the findings from a series of 488 children that the average age for the initial attack was 8 years but that the onset was usually at 7 years of age. On an average, one-fourth of the patients had their first attack before school age, in the case of polyarthritis usually at 5 years of age, in the case of carditis at 6 years, and of chorea between 7 and 8 years.

In a series of 696 children in New York (May Wilson, 1940), the highest incidence for the first attack was between the ages of 6 and 7, the first evidence of the disease appearing in about half the cases between the ages of 5 and 8.

Among 314 cases of juvenile rheumatism and chorea in Dublin,

Holland Clarke (1940) found that in 79.3 per cent of all cases the first evidence appeared between the ages of 5 and 11 years — between 5 and 10 years in 61.2 per cent of the cases with rheumatic fever, and between 6 and 11 years in 81.7 per cent of the cases with chorea — the peak ages of incidence being at 9, 9, and 10 years, respectively.

Edström (1935) found among his 850 cases of acute polyarthritis from the southern districts of Sweden that in most cases the age at onset was between 11 and 25 years, the peak being between 16 and 20 years of age.

While the above-mentioned publications point to the fact that rheumatic fever is a disease often occurring in childhood, and that the incidence is highest between 5 and 11 years of age with a peak at 7—8 years, most of them suffer from the disadvantage that they only deal with children and young persons up to the age of 16 to 18. There is, however, one investigation from an earlier period which embraces patients of a higher age, and that is a paper by Wyckoff and Lingg (1926), but the purpose of this was to study the causes of heart disease in general. (It was established that 90 to 95 per cent of all heart diseases of a rheumatic nature occurred before 50 years of age and more than half of them before the age of 30.) It is of great interest, therefore, to find a paper by Hedley (1940), from Philadelphia, in which he deals with rheumatic fever at all ages. In his material comprising 4 538 cases of rheumatic fever, rheumatic carditis, and chorea, he observed that the peak age of incidence lay between the ages of 5 and 9. The onset occurred before the age of 20 in 69.1 per cent of the cases with rheumatic heart disease, in 76.4 per cent of the cases with rheumatic fever and in 98.2 per cent of the chorea cases. The corresponding figures for cases where the first evidence of the disease appeared before 15 years were 57.8, 63.1, and 94.6 per cent.

In an investigation on rheumatic affections in patients treated at the hospitals in Cincinnati, Wedman and Wedman (1944) found that half the patients were under 30 and that in 89 per cent of all cases of chorea the patients were between 5 and 14 years of age.

Cohn and Lingg (1943), after a study of 3 129 cases of rheumatic heart disease with a fatal outcome at the New York Cardiac Clinics, including patients of all ages, reached the conclusion that the average age for the initial attack is 14.5 ± 0.3 years for males and 15.0 ± 0.3 years for females. (The difference, 0.5 ± 0.136, was considered to be statistically significant.) The onset occurs as a rule between the ages of 5 and 10 years, the peak age of incidence being at 8 years. It was found that by 15 years of age "70 per cent of persons afflicted have already acquired the disease".

As regards *the relation between the sexes*, Rolly (1920) states that among 3 101 cases (the majority of them over the age of 15) 1 587 were males and 1 514 females. Vaubel (1938) found that the distribution on the sexes is on the whole the same as in the whole population; after a survey of the literature he concluded that the range of variability was 47—53 per cent in both sexes. Findlay (1931) states that in his series of children in Glasgow the relation between boys and girls, in cases with arthritis alone, was as 1 to 1.1, while in cases where chorea had also been present it was as 1 to 2.5. He also found a difference between boys and girls as regards the peak age of incidence, which in cases with arthritis, he stated to be 7 years for boys and 10 years for girls. Kaiser (1934) gave the percentage figures for boys and girls (including cases with chorea also) as 46 and 54, respectively. Poynton, Paterson and Spence (1920) gave the relation of boys to girls as 2 to 3, but they, too, did not separate the chorea cases from the rest of their material. Fanconi and Wissler (1943) had exactly the same number of boys and girls, if the chorea cases were not included. For the latter, the relation was as 1 to 1.4. According to a survey worked up by Findlay, the proportion of boys to girls, in the case of chorea, varies between 1: 1.5 and 1: 2.8 in the various reports.

Wilson had 387 girls and 309 boys in a series of 696 cases of rheumatic fever with chorea, which according to her was not a significant relation.

Nordgren (1922) had twice as many girls as boys (218 girls out of 343 cases) among his chorea cases. In 367 patients with

another rheumatic disease the preponderance was slightly in favour of the girls.

In Edström's (1935) material from Lund (862 cases) 47.5 per cent were men and 54.5 per cent were women. (His material, however, is composed solely of cases of polyarthritis, some of which were possibly of the chronic type.)

In Hedley's opinion (1940), which he based on his observations in a large material of rheumatic heart affections (4 538 cases), the conclusions reached by others, namely, that the disease is a slightly commoner occurrence in females than in males (almost three-fifths of the cases were females), are in accordance with the facts.

In an endeavour to *summarize* the conclusions in the above-mentioned publications, it might be said that rheumatic fever, in the large majority of cases, occurs initially in childhood and adolescence, showing a predilection for the ages of 5 and 11 years, the highest incidence being at 7—9 years of age; possibly in the case of chorea cases, the peak age of incidence is $^1/_2$—1 year later. Chorea, in practically all cases, occurs before the age of 20 years; other forms of rheumatic fever can make their first appearance at higher ages also, although in the vast majority of cases the onset occurs in childhood or the early years of adolescence.

If chorea cases are included, the relation between the sexes seems to be fairly even; possibly, females are attacked to a slightly greater extent than males. Chorea, on the other hand, occurs predominantly in girls, the proportion of males to females being 1: 1.4—2.8 in various reports.

Annual and seasonal variations.

That the number of rheumatic fever cases varies from year to year is a fact that has long been known, and regional epidemics of the disease have even been mentioned. From the aspect of yearly and seasonal variations also, circumstances seem to vary

with the geographical position. As regards the conditions in Sweden, Edström (1935) found that in the case of acute polyarthritis there were distinct incidence peaks during the years 1904—1905 and 1915—1916, and again to a marked degree in 1929—1932, while especially few cases occurred in 1918 and 1924; in 1934—1935 there was a falling tendency. Similar variations are mentioned in reports from other countries and districts (see Edström).

The occurrence of the disease during different seasons seems, as mentioned above, to vary somewhat according to the geographical position. It is a generally accepted fact that the disease is more likely to occur during the colder months, but while most authors are in agreement here they also point out that it can occur at all times of the year, although with more or less pronounced variations. Thus, according to Findlay (1931), the greatest incidence of rheumatic fever in conjunction with chorea was during the months of September, October, and November, during which time 41.2 per cent of the cases of arthritis (out of a total of 160 cases) occurred; the complaint was least prevalent, in this material, from March to July, only 25.6 per cent of the arthritis cases starting in this period; the conditions were practically identical as regards the chorea cases (out of a total of 144 cases). In London, rheumatic fever seems to be more common in September and October, according to Osler (1920), while Voelcker (1913), on the other hand, mentions the months of May and November.

In Rochester, Kaiser (1934) found that the disease is more prevalent in the late winter and spring months (among a total of 1 085 cases) and least common during the summer and autumn.

May Wilson (1940) also elicited the fact that rheumatic activity is greater during the winter and spring than during the summer and autumn, and that the incidence of the disease is lowest in the summer, although there may be variations from year to year.

Stroud et al. established during an investigation in Philadelphia on 685 children with rheumatic fever, including cases with chorea and carditis, that of 816 attacks the largest number occurred in March, with a high incidence as well in February, April and

May; 46 per cent of the 816 attacks occurred in these four months. The lowest number of attacks were in July, August and September.

Sutton (1928) reported that in New York the peak of the curve is reached in April, while the bottom is in October.

In a report on 713 children with rheumatic fever in Minnesota during 1932—1935, Shapiro (1936) stated that the disease shows a high peak during the spring and late autumn, and is least prevalent during high summer.

Grenet (1937) in France, when studying 352 cases, found two periods when the incidence was high, January—February and June—July, while in August—December it was low.

In Germany, the disease is most prevalent in the spring months (Wilson, 1940).

Nordgren observed in 1922, in a material from Stockholm consisting of 343 chorea cases and 369 cases with other rheumatic manifestations between the years 1908 and 1921, that the largest number of cases occurred in October—April, with a peak for chorea in January—March and for the other rheumatic cases in October—November. Edström demonstrated in his investigation of rheumatic fever (no cases of chorea were included) that an incidence peak occurs during the winter months while the lowest point is found in late summer and autumn.

Summarizing, rheumatic fever shows fairly wide variation in incidence from year to year, with more or less distinct maximum and minimum levels over certain year-periods. This oscillation in the occurrence of the disease is not uniform as regards different countries and districts.

Although there is a considerable amount of discrepancy between different materials as regards seasonal variation, it can nevertheless be said that a fairly consistent characteristic of rheumatic fever inclusive of chorea is its predilection for the winter and spring months and its fairly low incidence during the summer and early autumn months.

Incidence of various major manifestations.

The three major manifestations of rheumatic fever now generally accepted are acute polyarthritis, chorea, and carditis, the two first often occurring to a greater or lesser extent conjointly with carditis. Carditis, however, may occur without any signs of arthritis or chorea being observable, although signs of these conditions may appear in connection with a recurrent attack or at a later stage in the course of an initial attack of carditis. Particularly in American publications, another major group of symptoms, "growing and joint pains," is often mentioned; these symptoms, in my opinion, are rather to be placed in a subgroup under arthritis, to indicate that they are an expression of a subclinical form of the arthritic manifestation, in those forms of rheumatic fever in which other signs and symptoms demonstrate clearly that a rheumatic affection is really in question. Particularly in the case of growing pains much doubt must be felt, since this symptom is often present without there being any other sure signs of rheumatic infection. It is only if more definitely acknowledged rheumatic manifestations such as chorea, carditis, or acute polyarthritis, appear during the subsequent course of the complaint that there would seem to be justification for suspecting that the original symptoms may have been rheumatic in origin, and even then we cannot always be sure. Growing or joint pains as an expression of a rheumatic affection must, in fact, even at the initial attack, occur in conjunction with other signs and symptoms of rheumatic fever before they can be interpreted as forming part of a rheumatic infection.

Regarding the frequency of these manifestations in different regions, there are many reports, a few of which will be mentioned here.

Findlay (1931) had a series of 701 cases in which the combinations of the various major manifestations up to the time of writing were as follows: 322 cases (45.9 per cent) with arthritis including growing pains, 166 cases (23.7 per cent) with chorea, 179 (25.5 per cent) with arthritis and chorea (in all of these carditis was present as well), and 34 cases (4.9 per cent) with carditis

alone. The following figures were given for the earliest sign of the disease: carditis, 42 cases (6.0 per cent); arthritis, 372 cases (53.1 per cent), and growing pains, 74 cases (10.6 per cent) — making together 63.6 per cent — and chorea, 213 cases (30.4 per cent).

In a survey of 545 cases of rheumatic fever in Dublin, Holland Clarke (1940) found 239 cases (43.9 per cent) with signs and symptoms of polyarthritis, 73 cases (13.2 per cent) with pains in the joints in the personal history but no objectively discoverable signs, 156 cases (28.6 per cent) of chorea, and 76 cases (13.9 per cent) of carditis without mention of chorea or polyarthritis in the history. (In addition, there was one case which he stated was "rheumatic erythema nodosum".) Unfortunately, it is not mentioned whether, in this system of grouping, it was a question of initial symptoms or not.

In a series of 1 181 children in Rochester, Kaiser (1934) found the frequency of the initial rheumatic manifestations to be as follows: 723 cases (61 per cent) of acute arthritis, 326 cases (28 per cent) of chorea, 140 cases (12 per cent) of carditis (primary), and 98 cases (8 per cent) of muscular rheumatism (or "growing pains"). The frequency of carditis in these groups was 67, 58, 100, and 43 per cent, respectively. In the arthritis and chorea groups there are included 106 patients who had both chorea and arthritis at one and the same attack, and these were consequently counted in each group.

Ash (1936) reported that in a series of 445 cases carditis was the initial manifestation in 74 cases (16.6 per cent), polyarthritis with fever in 257 cases (57.7 per cent), "growing pains" in 17 cases (3.8 per cent), and chorea in 97 cases (21.3 per cent).

Wilson (1940) stated that in her material there were 699 cases in which it was possible to establish the nature of the initial manifestation. Growing and joint pains, chorea, and polyarthritis each occurred in one fourth of the patients (25.6, 23.2, and 22.6 per cent, respectively), without being combined with other signs or symptoms. Pure primary carditis was found in 25 cases (3.3 per cent), and in addition to this she had a group of cases with "carditic murmur" (73 cases, 10.5 per cent). A

striking feature is the low incidence of carditis in these groups in connection with the initial manifestation, carditis only being found in 11.7 per cent of the patients with polyarthritis and chorea. Polyarthritis and chorea were present in conjunction in 22 cases (3.1 per cent). The remarkably low incidence of complicating carditis can perhaps be partly explained by the fact that only the initial manifestation is being discussed here.

If we divide up Wilson's material among the three major manifestations, polyarthritis, chorea (in both instances with or without carditis), and carditis alone, we obtain the figures 223 (31.9 per cent), 171 (24.5 per cent) and 96 (13.8 per cent), cases with cardiac murmur being included, and in addition to these we have a group of 30 cases (4.2 per cent) with polyarthritis and chorea (with or without carditis) as well as the group with "growing and joint pains," comprising 179 cases (25.6 per cent).

Summarizing, we find that chorea as a primary lesion of rheumatic fever is present in 20—30 per cent of all cases. Carditis as the sole major manifestation is stated by different authors to be present in, at the minimum, 5—6 per cent, and at the maximum, as a rule, in about 16—17 per cent of all cases. The remaining cases show more or less marked arthritic symptoms. The variation in the figures mentioned by different authors is in all probability due to differences in materials, as well as to varying standards for evaluation. To place cases with growing and joint pains in a special group has been suggested by some authors, especially in America, but in those cases of proved rheumatic fever in which these symptoms occur it would appear to be more suitable to place them in the group with arthritic symptoms.

Prognosis.

There is little mention of the prognosis of rheumatic fever in the older publications, and even where this aspect is mentioned the question of the late prognosis is scarcely touched upon. In

recent years, a change has been taking place in this respect, although the length of the observation period must in general still be regarded as too short to allow definite conclusions to be drawn as to the prospects for the future. It is only now becoming possible, because of the greater clarity that has been reached during the past twenty to thirty years regarding the signs and symptoms of rheumatic fever, to assemble a fairly large series of cases which fulfil the present-day requirements as to exactitude of diagnosis. It is often difficult, if not impossible, to pick out those cases in earlier publications which can be said to be cases of rheumatic fever according to the standards of the present time. Consequently, the conclusions that have been drawn from earlier publications must also be looked upon with a certain amount of reservation, and too much reliance cannot be placed on the figures reported. As a consequence of the above-mentioned circumstances investigators have not yet had at their disposal for any length of time a large number of cases of which it could be said with any great degree of certainty that they were undoubted cases of rheumatic fever, inclusive of chorea. The comparative shortness of the observation period has therefore been such that no far-reaching or final conclusions could be drawn. It is only the more recent publications that contain series of well-documented cases of rheumatic fever followed over a long period.

Mortality.

The average death rate according to various authors is 20 per cent (Fanconi and Wissler, 1943). Ash (1936) mentioned that among 401 cases in Philadelphia, followed for 10 years, the mortality was 22.3 per cent, this figure including also 5 children with terminal subacute endocarditis due to *Streptococcus viridans*. Fanconi and Wissler (1943), among their own 200 cases, had 27 deaths (13 per cent), but they estimated the figure to be higher as they had lost contact with some of the children. (No mention was made of the observation time.) In a series of 564 children in Rochester, Kaiser (1934) found an average mortality rate of 8.2 per cent during a 10 year observation period, but as a fairly

large number of these patients had only been followed for 3 years this figure must be regarded as too low. The death rate for the first 3 years during which all the patients were under observation was 5 per cent; among those who were under observation for a further period of 2 years (341 children) it was 1 per cent during these 2 years, while during the following 5 years 6 per cent (out of 180 children) died.

In an analysis of 1 000 cases of juvenile rheumatic fever, 979 of which had been under observation for at least 2 years (the mean observation time was 4.75 years), Schlesinger (1938) found a mortality rate of 12.3 per cent. Some of these cases had been under observation for 10 years, and the majority between 2 and 6 years.

Findlay (1931) has worked out an interesting table with regard to 257 fatal cases of rheumatic fever in which the age at onset was known. The material includes cases in which the initial attack occurred up to the age of 60. He found that of those contracting the disease before or up to the age of 5, 47 per cent had died within one year of the first manifestation and 97 per cent within 10 years. "The proportion of deaths not only during the first year after onset, but also during the first ten years after onset, steadily falls to the age-period of onset of 16 to 20 years. The smallest number of deaths and the longest duration of the cardiac infection occurred when the rheumatism was contracted between 20 and 30 years". Among 619 cases of rheumatic fever observed by Findlay in Glasgow between 1914 and 1929, there were 154 deaths (24.9 per cent). When the cases were divided into age-groups of 4 years according to the age at the initial attack it was found that in the 2—4 year age-group 18 had died (41.8 per cent), while in the following age-groups, and up to the age of 20, the corresponding percentage figures were 27, 20.5 and 23.8. The number of cases in the lowest age-group (43 cases) was too small, however, to permit definite conclusions to be drawn.

Martin (1941) found a mortality of 29.9 per cent (431 cases) in a series of 1 438 children with rheumatic heart diseases treated during a 20 year period at convalescent homes in New York. Of these 431 cases, 333 had "a history of a primary attack of

rheumatism," and 142 (42.6 per cent) of these 333 died within 5 years of their initial attack; in other words, they died before puberty, since the mean age for the initial infection was 6 to 8 years. Eighty-seven per cent of the deaths occurred before the age of 20. This investigation, however, only deals with cases undergoing care in a convalescent hospital, and as these patients constitute a selected group, being survivors of the initial attack considered to have some chance of living a reasonable time, the value of the results is rather limited. Furthermore, a number of patients with congenital heart disease were included in the total number of cases.

Stroud and Twaddle (1940) had 144 (21 per cent) deaths in a group of 685 children treated at the Children's Hospital, Philadelphia, during a 15 year period (1922—1937). But the authors themselves stated that this figure was "unnecessarily high, since during the first few years of the institution the wrong type of cases was accepted". After limiting their calculations to children treated from 1927 to 1937 they found a mortality of 14.8 per cent.

Duckett Jones (1938) stated that of the 1 000 patients with rheumatic fever treated since 1921 at the House of the Good Samaritan in Boston, 24.2 per cent had died after an average period of 10 years (mean age at onset, 8 years; mean age at last observation, 18 years). He gives no detailed analysis of these 242 cases, however.

Coombs (1924) found, in a series of 253 patients with rheumatic heart disease in Bristol, that 21.4 per cent had died within a period of 10 years after the first evidence of the disease.

Grenet and Joly (1938) reported that in 352 cases (112 of which were chorea) they had a mortality rate of 17.4 per cent during a period of 10 years, while among cases with clinically demonstrable heart affection the mortality was 23.9 per cent.

In a previously mentioned investigation carried out by Wilson (1940) it was found that 131 children out of 717 (18.2 per cent) had died, and death among these was due to cardiac disease in 117 cases (16.3 per cent). The mean age of death was thirteen years. The mean duration of the disease in 112 deceased patients, was six years. Thirteen patients, or 2 per cent, out of 702 died

before the end of the first year's illness, 61, or 10.4 per cent, out of 587 patients where the course was known for 5 years died within this period, and 102, or 29 per cent, out of 348 patients where the course was known for 10 years died before 10 years were over. From the standpoint of the mortality risk, and for purposes of comparison, the two last percentage figures seem too high, since all cases dying within 5 and 10 years, respectively, of the onset were apparently included in the respective groups, while corresponding non-fatal cases contracting the disease at approximately the same time as the last-mentioned patients who died were not included in the total figure until after an observation period of at least 5 and 10 years, respectively. From the report by Coombs (1924) concerning 374 children observed for the first time before World War I, Wilson deduced corresponding figures for the number of deaths within 1, 5, and 10 years, viz. 4.0, 12.2, and 17.2 per cent.

In an investigation on 3 129 cases of rheumatic heart disease which were followed to the time of death Cohn and Lingg (1943) found that 50 per cent had died within 9 years of the onset of the disease, 25 per cent survived for more than 17 years and 10 per cent for 30 years or more. From their material they were also able to deduce that "when the disease begins in childhood, 69 per cent survive childhood, 35 per cent survive adolescence, 18 per cent reach the age of 30, and 5 per cent go on beyond the age of 45. When the disease begins in adolescence 85 per cent survive this age period, 55 per cent reach the age of 30 and 21 per cent the age of 46 or more. When the onset is in the twenties 23 per cent and when it is after 30, 44 per cent survive the age of 45." Unfortunately, only the cases ending in death are described in this investigation, which was conducted on the basis of records from some 12 000 patients who received treatment over a certain period (15 years?) at the New York Cardiac Clinics. The figures are therefore based solely on the patients who died, and furthermore they only include patients with rheumatic heart disease and not cases without any demonstrable signs of it. These facts should be borne in mind when the figures are being evaluated.

That the figures for the mortality rate show such variation is probably to be ascribed to the fact that the various authors

have used different criteria for the diagnosing of rheumatic fever, with the result that the less severe cases have not been included or even, perhaps, not treated at the hospitals in question. It is usually impossible to form a clear idea of which cases have been included in the group in question by studying the different reports, and direct comparisons between them are therefore impossible. Thus, Martin's material, mentioned above, included only children with rheumatic heart disease, a fact which, in consideration of the known predilection of the disease for, and its frequent deleterious effect on, the heart, must involve a risk that the mortality rate in this group, composed especially of cases with demonstrable heart affection, will be unusually high.

Another reason for the variation in the mortality figures of different authors is, of course, the variation in observation times in the various investigations, as well as the variation between different geographical areas.

A remarkable feature is, also, that Coombs, and Wilson, for instance, give a very low mortality rate for the first year (4 and 2 per cent, respectively) and a fairly high rate (17.2 and 29 per cent, respectively) after an observation period of 10 years, while Findlay on the other hand, in his fatal cases, mentions that the rate is already high for the first year following the onset (24.5—47.0 per cent for various ages in childhood). (Cf. also the mortality figures stated by Kaiser.)

To sum up, rheumatic fever shows a very high mortality rate, varying between approximately 12 and 30 per cent, according to the different reports, after a mean observation period of ten years. In some publications the majority of the deaths are said to take place during the last half of this period; in others (Findlay) the vast majority seem to occur within one year of the onset of the infection.

Recurrence rate.

A strong tendency to recur has for long been considered to be one of the most characteristic features of rheumatic fever.

As already mentioned (page 15), this tendency is regarded by many as so typical that it can be used as a decisive sign for the diagnosis, particularly in uncertain cases. As stated earlier, recurrent attacks can be expected to occur in, on an average, 70 per cent of the cases (from 50 to 85 per cent, according to various authors). In these calculations, however, the total number of patients who had a primary attack is sometimes included, whereas still higher and more adequate figures for the recurrence rate would be obtained by deducting, instead, the cases dying at the initial attack and calculating the percentage of recurrences in relation to all cases surviving the initial attack.

Kaiser (1934) found, in a series of 564 cases of juvenile rheumatic fever followed for 3 years (341 were observed for 5 years and 180 for 10 years or more), that 49 per cent of the children had one or more recrudescences during the first 3 years. During the period from 3 to 5 years 40 per cent of those followed had one or more recurrent attacks. It was not until the period from 5 to 10 years following the initial attack was reached that the recurrence rate dropped to 25 per cent. Unfortunately, there is no information as to how many cases, observed for 10 years, had no recurrences, nor are there any other statements regarding the occurrence of recurrent attacks.

During a mean observation period of 8 years (up to a total of 15 years), in which 488 cases of rheumatic fever were followed, Roth et al. (1937) noted that at least one recurrence occurred in 68 per cent of the patients; in the case of chorea the figure was 74 per cent. In 73 per cent of the cases with recurrences (64 per cent of those with polyarthritis, 85 per cent of those with carditis, and 81 per cent of those with chorea at the initial attack), the first recurrence occurred within 3 years at the latest. Forty per cent had at least 2 recurrent attacks, and nearly two-thirds of these occurred within 4 years of the initial attack.

Edström (1935) submitted 694 cases of polyarthritis from the years 1911 to 1933, collected in the province of Malmöhus (Malmöhus län), Sweden, to a follow-up examination in 1934—1935. All ages were represented, but not very many of the patients were actually in what is generally considered to be the age of child-

hood. Edström's result was that about 50 per cent had one or more recurrent attacks. Of these recurrences, 27 per cent occurred during the first year following onset, and the vast majority of the cases had their first recurrence within 10 years of the initial attack. Only in 12 per cent of the total number of cases was the first recurrence later than 10 years after the first appearance of the disease (only 6 per cent of the total first recurrences occurred after the lapse of 10 years). Edström also reported that the risk for a recurrence lessens as the age at onset increases, and is greatest when the initial attack occurs in the years of childhood and adolescence. It should be pointed out, however, that among these 694 patients there were apparently a number with chronic polyarthritis (about 21 per cent). Further, the lower ages are poorly represented in this material and there are few cases below the age of 10.

Wilson and Lubschez (1944) studied the risk of recurrence relative to the age-period between 4 and 16 years in a group of 499 cases of rheumatic fever, the average observation time being 8 years. Within this group of 499 cases there was another one comprising 345 patients who reached the ages of 17 to 25 years, the mean observation time here being a further 6 years (thus, a total observation time of 14 years). In the former group, 31 per cent had no further recurrence of the disease, and in the other group there were no recurrences in 87 per cent in the following 6 years. The average recurrence rate for different ages was found to be 25 per cent for patients between the ages of 4 and 13 years, 8.7 per cent for those between 14 and 16 years of age, and 3.7 per cent for patients of 17 to 25 years old. The recurrence rate was highest at 7 years of age and after the age of 13 there was a significant downward trend in the risk of a fresh attack. "For a random sample between the ages of 4 and 16 years, the average risk of recurrence was 20.6 per cent.

The interval of time elapsing between recurrent attacks for patients between the ages of 4 and 16 years was also found to be an important factor in the risk of future recurrence. The risk of recurrence during the year immediately following a major episode was 38.7 per cent compared to 11.2 per cent in the year

immediately following one year of freedom from recurrence. The average rate of recurrence within one year or more after a major rheumatic episode was twice as great as that following at least one year of freedom and three times as great as that following at least two years of freedom from symptoms, 20.6 per cent, 10.7 per cent and 6.6 per cent, respectively."

The risk for a first recurrence after onset was calculated by these authors as being, on an average, 20.6 per cent, while the risk of a second recurrent attack for those patients who had already experienced one recurrence was 18.6 per cent and the rate for a third recurrent attack for patients who had experienced two recurrences was 22.1 per cent. The risk of future recurrences was thus not influenced by the number of recurrent attacks. The only factors which were found to influence the future recurrence rate were age and the interval of time elapsing since the last attack.

According to Cohn and Lingg (1943), the largest number of recurrences occur between the ages of 5 and 14 years. (In a series of 12 000 patients with rheumatic heart disease they selected 3 129 patients who died during a certain space of time, and among the last-mentioned cases 75 per cent had had one or more recurrent attacks.) The recurrence curve for the 3 129 cases showed a definite drop after 11 to 12 years and after the age of 20 years it remained fairly constant. They found, also, that the risk of recurrence diminishes in adolescence, a finding which, in view of the predilection of the initial attack for 7 to 8 years, coincides with about 5 years after the onset. Unfortunately, this investigation was only concerned with patients already dead; if the cases which survived had been included the figures would undoubtedly have been very different.

In general, it would seem to be accepted that the more recurrences of rheumatic fever a person passes through the greater is the probability that a permanent heart affection will develop. Findlay (1931) found, however, as the result of an exhaustive study of 108 closely observed cases about which everything was known from the beginning of the disease, that the heart affection always makes its appearance during the course of the first and second attack and never after the second attack. In a series

of 643 patients with rheumatic fever he noted that among those who had had one attack only there were signs of cardiac affection in 67.5 per cent after arthritis and in 45.2 per cent after chorea. Among those who had had two attacks, heart affections were present in 63.9, 60.9 and 54.2 per cent, respectively. In patients who had had 7 attacks he found a 100 per cent incidence of heart disease which, in his opinion, had already developed during the first or second attack.

Summarizing, about 70 per cent (50—85 per cent) of all patients contracting rheumatic fever sooner or later have one or more recurrent attacks. The risk for a recurrence is greatest during the first 1—3 years following the initial attack. The recurrence rate seems to fall after adolescence. With regard to chorea, the risk for a recurrence of this or some other major manifestation of rheumatic fever seems to be greater than it is for other forms.

Prognosis as regards the heart.

Rheumatic fever may without doubt be regarded as the disease which gives rise to a very large part, perhaps the majority, of the heart complaints, in particular the valvular lesions, occurring during adult life. Those who have made a close study of this disease are all agreed, also, that the cardiac affection and the degree of the heart damage remaining after an attack of rheumatic fever is the deciding factor as regards the future prospects for the patient. It would also seem to be generally agreed that one has no right to draw final conclusions regarding the condition of the heart during or directly after an attack, since the signs of a valvular affection not infrequently change during the period following the attack, tending sometimes to disappear and sometimes to change to new or more pronounced signs.

Thus, at a follow-up carried out by John and Nobel (1923) on 78 children treated at the children's clinics in Vienna, where the observation time was from $5\,^1/_2$ to $18\,^1/_2$ years, it was found

that 11 patients (26 per cent) who had presented signs of cardiac involvement directly after the attack of polyarthritis had a normal heart at the follow-up. A normal heart was found at the follow-up in 42.8 per cent of the 42 patients who had had polyarthritis alone. Among the 21 cases with chorea, 10 patients (47.6 per cent) were found at the follow-up examination to have absolutely normal hearts. The others had involvement of the heart in some form. All the 6 cases with a combination of chorea and polyarthritis later presented signs of heart damage. The number of cases under observation is unfortunately too small to allow any far-reaching conclusions to be drawn.

Table 1.

Incidence of heart disease following rheumatic fever.

Observer	Total cases	Demonstrable rheumatic heart disease	Potential rheumatic heart disease
Wilson, Lingg and Croxford (New York, 1928)	416	79.5 %	20.5 %
Findlay (Glasgow, 1932) . .	644	66.4 %	33.6 %
Kaiser (Rochester, N. Y., 1934)	1,240	64.0 %	36.0 %
Ash (Philadelphia, 1936) . .	445	66.1 %	33.9 %
Schlesinger (London, 1938) .	1,000	74.2 %	25.8 %
Bland and Jones (Boston, 1938)	1,000	68.6 %	31.4 %
Average	4,745	70.8 %	29.2 %

From Bland and Jones (1939).

According to a survey (see table 1) worked up by Bland and Jones (1939), the heart can be expected to be involved during the course of a rheumatic infection to a sufficient extent to become clinically demonstrable in about 70 per cent of all cases. It seems to be generally believed that the remaining 30 per cent of rheumatics also have mild cardiac damage during their infection, although this may be so slight that it is undetectable by present methods of clinical observation.

Bland and Jones described in 1939 an investigation on 1 000 patients with rheumatic fever who had been under observation for a period of 10 years at the House of the Good Samaritan in Boston. In this series, there were 314 who, on recovery from their initial rheumatic infection, were left without demonstrable damage to the heart. Among these, during the following 10 year period, characteristic signs of cardiac involvement appeared in 79 patients (25 per cent). Approximately two-thirds of these 79 patients (55 patients), however, had had a recrudescence of rheumatic fever or chorea, while the remaining 24 did not show signs of active rheumatic infection during the subsequent course. The authors themselves, however, consider that in the majority of these patients there was some subclinical form of recurrence.

Duckett Jones (1938), among his 1 000 children with rheumatic fever, had 328 patients who had no definite signs of cardiac involvement when discharged from the hospital. Out of these 328 patients, who were observed for a mean period of 10 years, 251 continued to be free from heart deformity; the remaining 71 all developed cardiac deformity of greater or lesser severity, in 5 cases with a fatal outcome, but in approximately half of these 71 patients the cardiac involvement had arisen in connection with a recrudescence of the disease. Among the 672 patients with established cardiac involvement at the time of their discharge from the hospital, the physical signs of this heart damage were found to have decreased in 53 cases while in 66 cases they had wholly disappeared.

In an investigation on chorea, Jones and Bland (1935) picked out a group of 134 cases in whom chorea was the sole manifestation of rheumatic fever from among a total series of 482 rheumatic fever patients, who had also had chorea and who had been under observation for a mean period of 8 years. An interesting feature of this group was that only 4 patients (3 per cent) had signs of cardiac affection. On the other hand, as soon as other rheumatic manifestations sooner or later occurred, 73 per cent of the chorea cases (out of 348 cases) also showed cardiac involvement, a figure which agrees well with the cardiac involvement in another group with rheumatic fever alone, without symptoms

of chorea (518 cases), where cardiac affection was present in 86 per cent. Contrary to this finding, Ash (1936) observed in a group of 445 children, 93 per cent of which had been followed up for more than 2 years, and where the mean observation time was $7\,^1/_2$ years, that valvular involvement was present in 13.3 per cent of her cases of pure chorea without other rheumatic manifestations (45 cases of pure chorea in a total of 153 chorea cases); the mortality in this group was 4.4 per cent. In the patients who had both attacks of chorea and polyarthritis, on the other hand, 71.5 per cent had valvular lesions and 17.5 per cent had died. In the whole group, 22.3 per cent were dead, and in 66.1 per cent there was cardiac involvement.

Grenet (1937), who followed 352 rheumatic fever patients (112 of whom had chorea) for a 10-year period, noted that 72.7 per cent had cardiac affection, which had led to permanent cardiac damage in 65.2 per cent (with a fatal outcome in 23.9 per cent).

Schlesinger (1938) submitted a report on 1 000 cases of juvenile rheumatic fever in London followed up for from 1 to 10 years after the initial attack, which had occurred during the years 1927—1935. The mean observation time was 4.75 years. Two-thirds of the patients were observed for 4—10 years. Of the total number, 12.3 per cent had died. Of the surviving 877 children, 572 showed heart affection (568 valvular lesions and 4 myocarditis alone). Mitral insufficiency was present in 344 (39.2 per cent), mitral stenosis in 166 (18.9 per cent), aortic insufficiency in 11 (1.1 per cent), and aortic stenosis in 1 (0.11 per cent). In 46 cases (5.2 per cent) there was combined mitral and aortic affection.

Edström (1935) had 323 cases between the ages of 4 and 20 years, treated for acute rheumatism of the joints between 1911 and 1933 at Malmö and Lund, Sweden. Of these, 240 (74 per cent) were found to have cardiac involvement at a follow-up investigation during 1934 and 1935. As already mentioned, however, this material included a number of cases of chronic polyarthritis, and this figure therefore cannot be regarded as wholly reliable.

Holland Clarke (1940), who reported from Dublin on 545 cases of "juvenile rheumatism," found cardiac affection in 49.6

per cent of 231 cases of rheumatic fever (without chorea), while the corresponding figure for 156 cases of chorea was 27.6 per cent. Among 11 cases of post-scarlatinal rheumatism he had 72.7 per cent with valvular damage. Unfortunately, in this report, there is no mention of the length of time from the initial attack to when the cardiac involvement was established (in connection with the hospital stay?).

In a series of 673 children observed by May Wilson in New York during the 20 year period 1916—1936, where the mean duration of illness was 8 years, cardiac involvement was found in all except 4 cases. In the 325 patients under observation either before or within one year after the onset of the disease it was established that "cardiac involvement was recognized within the year of onset of rheumatic fever or in the same year in 85 per cent of the cases". The heart lesions found at the last examination were distributed as follows on the 582 cases surviving at that date:

		Percentage of total 673 cases	Percentage of the 582 survivors
Mitral insufficiency ..	281 cases	41.9	48.3
Mitral stenosis with or without insufficiency	217 »	32.3	37.2
Aortic and mitral lesions	36 »	5.4	6.2
Enlarged heart.....	29 »	4.3	5.0
Undetermined.....	15 »	2.2	2.6

Ninety-one (13.5 per cent) of 673 patients had died and in 4 cases (0.6 per cent) there was no heart disease.

Wilson also divides her material into patients in whom active carditis was established during the period of illness (246 cases), and patients in whom, in consideration of the cardiac involvement detected at a later stage, a subclinical carditis must presumably have been present (427 cases); the latter cases, for the sake of convenience, were called cases "without carditis". In point of fact, an examination of different types of rheumatic fever revealed in her material, that "active carditis was the only manifestation

of prognostic significance". It was found that among 246 children with "active carditis" 36.2 per cent died, and approximately 50 per cent, although still living, showed signs of more or less severe cardiac damage. Among the remaining 427 patients (about two-thirds of the whole series of 673 patients), only 2 had died, and the cardiac affection was much less severe. In these two groups there was no significant difference as regards the degree of cardiac affection, whether the course was characterized by growing and joint pains, chorea, or polyarthritis. Nor did an attempt to find a difference between the patients who only had chorea during their illness and those who had other symptoms in association, or those with other symptoms only, yield any significant result. She considered, also, that the risk of mitral stenosis or aortic insufficiency rose with the number of attacks. As regards mortality, the figure rose from 20 per cent, in cases of active carditis with only one attack, to 64 per cent after 4 attacks or more.

In a series of 1 000 cases which had been observed for an average period of 10 years (mean age at onset, 8 years, mean age at last observation, 18 years), Duckett Jones (1938) noted that 24.2 per cent were dead, 427 (42.7 per cent) had definite heart disease, and 310 "potential heart disease" (21 cases, insufficient information). Of the 427 patients with cardiac damage, 287 patients (67.2 per cent) were not limited at all as regards the physical activity, while in 119 cases (27.9 per cent) there was moderate limitation, and in only 21 cases (4.9 per cent) was there any great limitation of activity. Out of the total 1 000 patients, 597, or nearly 60 per cent, were able to carry on a normal physical life after an average period of 10 years from the initial attack.

Pericarditis is not infrequently an initial manifestation in rheumatic fever (Helmreich, 1935), but it occurs more often as a complication or a sign of more serious carditis. Pericarditis is the commonest cause of death (Helmreich). It occurs in greatest numbers in children of 7 to 11 years of age (Glanzmann, 1935). It is all too seldom diagnosed in the living, as is proved by the fact that all autopsy material shows a much higher percentage of this form of cardiac lesion than do the clinical data. Coombs (cited from Vaubel), for instance, found pericarditis in 50 per cent

of his autopsy cases, whereas the clinical signs of it were only observed in 13 per cent.

Schlesinger (1938), in his series, had 62 patients with pericarditis (6.2 per cent of 1 000 cases) whom he had followed for an average period of 6 years. Thirty-one of these died (50 per cent), 24 (39 per cent) were alive and in good health, and 7 (11 per cent) were more or less cripples. Two-thirds of those who died succumbed as a direct consequence of the acute attack.

Findlay (1931) states that pericarditis is seldom associated with chorea. Among his pericarditis cases (69 patients) he found that this complication coincided with an attack of chorea in only 4 cases, in one of which the pericarditis preceded the chorea. In 28 cases (40.6 per cent of the total number of pericarditis cases), on the other hand, the pericarditis followed on an attack of arthritis. In 6 out of the 69 pericarditis cases (9.8 per cent of 701 cases) pericarditis was the primary manifestation. Forty-five of the cases were girls and 24 boys. The average age of onset of pericarditis was 8.5 years, and the highest rate of incidence was at 7—11 years of age, but on the whole the age distribution showed the same features as in the total series of cases. There was a recurrence of pericarditis in 6 cases. Of the 69 cases, 40 died (63.4 per cent), 33 at the acute stage and the other 7 during the following 7 years. In only 2 of the survivors were there no signs or symptoms of cardiac disease, 12 had cardiac disease without subjective symptoms, while 15 had subjective discomfort as well. In other words, 79.7 per cent of the children with pericarditis were either dead or more or less severely "incapacitated".

Edström (1935) reported that among his 850 cases, pericarditis had been diagnosed clinically on some occasion in 71 (+ 1?) cases (8.4 per cent). In addition, there were 14 cases where the condition was discovered for the first time at autopsy. Thus, in all, 85 cases, or 10 per cent of the whole material. Half of these occurred before the age of 20. Thirty-six (approximately 50 per cent) of all the pericarditis cases had died.

Among 248 cases of polyarthritis and 198 cases of chorea in a Stockholm series, Friedländer (1945) found pericarditis in 23 and 9 cases, respectively; 7 and 2 cases, respectively, died (probably

in connection with the actual attack? No follow-up carried out on his series?).

Pleurisy not infrequently occurs in association with pericarditis; in these cases it is often left-sided and as a rule would then seem to be secondary to the pericarditis, but in a fair number of cases it may also be bilateral. It is of very short duration, about 8 days in some cases, but it usually lasts for 2 to 3 weeks, and often leads to adhesion of the visceral and parietal pleurae (Glanzmann, 1935).

The fact that nodules often occur in association with pericarditis is stated by Findlay to be a proof of the gravity of the infection. He observed these nodules in 29 per cent of his pericarditis cases, as compared with about 10 per cent in the whole material.

The publications on the *electrocardiographic changes*, which persist for a fairly long time after a rheumatic infection, are few in number. The investigations dealing with electrocardiographic abnormalities in rheumatic fever are nearly all concerned with the condition present during an active, progressing infection. In those papers mentioned above where the prognosis of rheumatic fever is discussed, there are, as a rule, no statements whatsoever regarding possible persisting abnormality in the electrocardiogram.

On the other hand, the literature dealing with changes during the acute stage of the disease is extremely rich. As already mentioned, it is now fairly generally agreed that practically all patients with rheumatic fever display at least mild signs of carditis at some time during the course of the disease, but that these signs are often not detected if the patient is not regularly and consistently examined, especially by means of the electrocardiograph. Thus, Cohn and Swift, as early as 1924, demonstrated by means of frequent electrocardiographic examinations at short intervals (in 37 cases an electrocardiogram was made every twenty-four hours, making a total of 2 591 electrocardiograms), that 35 of the 37 patients had changes in their tracings during their attack of rheumatic fever; these changes were in the form either of an increase in the auriculo-ventricular conduction time, alteration in the ventricular complex, or changes in the cardiac rhythm.

In an investigation reported by Reid and Kenway (1928), these changes were to be seen in 92, 80, and 34 per cent, respectively.

In a study of 181 patients with chorea in all attacks, a total of 314 electrocardiograms having been made, Heffer (1941) found abnormality in the electrocardiographic tracings in not less than 39 per cent, while there had never been any other signs of heart disease.

A study of electrocardiograms from persons with "quiescent rheumatic disease" reveals, according to Shapiro (1930), the same changes as those seen in the acute stage, but they are less frequent. The changes in his series (119 children and young adults) varied in approximately the same way as in the acute cases. On the other hand, no changes were observed in control cases examined at the same time. The commonest findings were the absence of an isoelectric period between the R or S wave and the T wave, and the marked variability in the S—T or R—T intervals from time to time. The paper does not make clear, however, what the author means by saying that the disease was in the quiescent stage; he only states that the children were allowed to go to school.

May Wilson (1940) says that "the distinctions between electrocardiographic abnormalities tabulated for active and inactive rheumatic heart disease are arbitrary, since any or all of these findings could be present in either stage". Isolated findings on the electrocardiogram are often of lesser significance than the progressive or regressive nature of the changes. According to the same author, the following electrocardiographic abnormalities are found in inactive rheumatic heart disease, viz. axis deviation, abnormalities of contour, notched, peaked, broad P, splintered QRS, low voltage, bundle branch block, and auricular fibrillation. No further information is given, however, as to the distribution of these changes.

Summarizing the prognosis of rheumatic heart disease, it should be pointed out, to begin with, that despite the fact that the heart is found to be involved in nearly every case, especially if frequent electrocardiographic examinations are carried out,

the condition is only demonstrable clinically in about 70 per cent of cases. Although the cardiac damage disappears entirely in a few cases, it persists in the majority, and in most cases may be expected to become more pronounced as time passes. Even in patients who show no clinical signs of cardiac involvement at the initial attack cardiac disorder seems in time to develop, even if there are no subsequent recurrent attacks (unless these possibly occur in a subclinical form).

The commonest form of valvular lesion appears to be insufficiency of the mitral valve (34—49 per cent) and after that, as a close second, mitral stenosis or a combination of these two defects (19—37 per cent). Pure aortic defects, on the other hand, are much rarer, apparently occurring only in approximately 1 per cent, while a combination of mitral and aortic deformity seems to occur in 5—6 per cent of surviving patients. No heart defect is found in 0.6 to 23 per cent, according to various investigators. (The variation between the different forms of heart defect would seem to be due to the different criteria for the diagnosing of cardiac disorders employed by the various authors, as well as to variation in the length of the observation period from onset to follow-up examination.)

The incidence of cardiac affection seems to be much lower (3—13 per cent) in cases where chorea is the major manifestation of every attack. For all types of rheumatic fever there are, of course, various transition forms between mild and severe cardiac damage.

The appearance of pericarditis in these cases with carditis darkens the outlook for the future to a very considerable degree, and constitutes the commonest cause of death in patients with rheumatic fever, about 50—65 per cent of pericarditis cases succumbing. It sometimes happens, however, that a patient with

established pericarditis does not later yield evidence of cardiac disorder.

Electrocardiographic changes of various types are observed in nearly every case of rheumatic fever, and in some patients these become permanent, although they usually seem to recede to a certain extent. The publications dealing with persisting electrocardiographic changes are, however, scanty.

* * *

In general, the literature on the prognosis of rheumatic fever by various authors seems to be defective in some respects, such as, too small a material as the basis for evaluation or too few cases from the original material available for a follow-up examination; prognosis determined only in relation to rheumatic heart diseases with exclusion of patients without cardiac involvement; no discrimination between patients with acute rheumatic fever and patients who from the beginning had a chronic form of polyarthritis; omission of cases with chorea alone; inclusion in the total number of cases of various uncertain cases with such affections as "growing and joint pains"; and too short an observation time between onset and follow-up examination. Further, in some investigations, special stress has been laid on certain aspects as regards the prognosis while other quite important ones are disregarded, the value of the publication being thereby reduced to a certain extent. Besides this, the various authors' cases of rheumatic fever are collected from several different hospitals and districts, which causes still greater discrepancy as regards diagnosis and treatment in the same material. In Sweden and the other Scandinavian countries, no report has yet been published on the prognosis — particularly the late prognosis — of rheumatic fever taking into consideration the above-mentioned sources of error. The present investigation was planned with a view to filling this gap in some measure, especially with respect to Swedish conditions.

THE AUTHOR'S INVESTIGATION.

CHAPTER I.

The material.

Its composition and limitations.

During the years 1922—1940, 689 children[1] received treatment at the Children's Hospital, Gothenburg, for acute rheumatic fever (inclusive of chorea), some of them on two or more occasions. The object of the present investigation was to use this material, supplemented with a follow-up examination, in order to form some idea of the prognosis of this disease in a uniform group of cases restricted to one definite district in Sweden. The material was assembled by studying the case records with respect to all patients cared for at the hospital for rheumatic fever or one of its synonyms during the period in question. No cases of the primarily chronic polyarthritis or Still's disease type, nor of infectional arthritis of varying etiology, were included. For the sake of completeness, cases originally diagnosed as arthralgia were also considered, and in some instances these patients were examined or merely asked to fill in the questionnaire mentioned further on in this report; nothing was obtained, however, which could indicate that they had at any time had rheumatic fever. These cases were therefore not included in the present investiga-

[1] Extracts from the records of all these patients as well as all other information obtained, together with the results of the follow-up investigation (including electrocardiograms), are obtainable in Swedish from the library of Norrtull's Hospital, Stockholm. X-ray films taken at the follow-up investigation, as well as the results of the roentgenographic examinations made during the active stage of the disease are available for examination at the Children's Hospital, Gothenburg, where the original case records are also to be found.

tion. In order to be sure that every case with rheumatic carditis without arthritic symptoms would be included in the series, all case records where the diagnosis was vitium cordis, endocarditis, myocarditis or pericarditis were studied, and relevant cases were taken in among the other rheumatic fever cases. These measures would seem to guarantee that no cases treated for rheumatic fever at the Children's Hospital during the period in question were left out.

As the Children's Hospital in Gothenburg is the only hospital for children in that city, it can be assumed that practically every case of this disease, among children requiring and receiving hospital care, is included in the series to be discussed here. In view of the fact that the very serious course not infrequently taken by this disease was known even at an early stage, it can be expected that the vast majority of cases coming to the notice of physicians were sent to the Children's Hospital for treatment. As regards children with very mild forms of the disease, it is, of course, probable that in some instances it was never considered necessary to seek medical attention, although in these cases also, the fear of the prolonged disability which, as most people know, is associated with diseases of the joints probably caused the majority of them to visit a doctor in time, as the result of which, in all probability, they were sent to the Children's Hospital. Although a few cases of chorea may have been cared for in the patients' homes, the character of the disease can be said to be such, that medical advice was sought in practically every instance, and that almost as many needed hospital care. Only in the case of the milder forms of carditis which arose in connection with acute infections or other such conditions is it without doubt very probable that a physician was never consulted, and that consequently the condition was never diagnosed. In cases where carditis appeared during medical treatment or where the condition was suspected during a medical examination, the patients were, with a high degree of probability, remitted to the hospital, if for no other reason than to have the diagnosis confirmed by means of the facilities of the hospital. It is therefore perhaps not incorrect to presume that the majority of children in Gothenburg with

carditis alone were also cared for at the Children's Hospital, in consequence of which they have also been included in my series of cases. The material is thus to be regarded as fairly representative, and of a suitable nature to serve as a basis for a prognostic study.

Thus, the original material is based on all the hospital case records relative to the above-indicated patients. As the Children's Hospital was under the charge of one and the same head physician during the whole of the period under review, and as all the records contain notes as to the state of health and physical condition, as well as the laboratory findings, for each week of the hospital stay, and additional comments in the case of more marked changes in the general picture, it was possible to obtain a fairly clear idea of the whole course of the disease in each individual case. A fairly large proportion of these patients (in particular those who contracted the disease during the latter half of the period 1922—1940) were also under observation, after they were discharged, at the out-patient department of the hospital. Frequent, thorough examinations were carried out at this department during the period immediately subsequent to the patients' discharge, their number and thoroughness being, of course, gradually reduced as time passed. Further, children contracting the disease during more recent years have had the advantage of a continuation of treatment at the "Broström Convalescent Home," an establishment recently opened in Gothenburg for rheumatic subjects.

The follow-up investigation.

Its plan and effectiveness.

All cases except six in the above-mentioned series were asked to fill in a questionnaire; in the case of very young patients, of course, the parents were responsible for the answers. The six exceptions were two men who have been at sea for several years, three women living abroad, in Norway, America, or China, and one woman who could not be traced, despite every effort to do

so. The patients were asked to reply as fully as possible to a number of questions regarding their present and past state of health, especially during the period following their treatment at the Children's Hospital, and to state clearly whether they had had any recurrence (or symptoms likely to suggest a recurrence) of the rheumatic disease, or any symptoms indicating the presence of heart trouble. Information as to other members of the family and as to whether the patients had done their compulsory military service was also requested. Besides this, all patients still living were asked to come to the Children's Hospital in Gothenburg (in a few cases where it seemed suitable, to Norrtull's Hospital, Stockholm) to have their present condition examined. In the case of patients who had died, as full information as possible regarding the place and cause of death was obtained from the Census Bureau, Parish Registers, and Public Health Offices. In addition to this, the parents or nearest relative of patients who had died were requested by letter to furnish information regarding the person in question and his family. In cases where it was reported that treatment had been received at another hospital than the Children's Hospital the desired information was obtained from the case records of the hospitals in question, which were kindly placed at my disposal by the respective head physicians.

The follow-up investigation was begun at the end of March, 1944, and completed for the most part during the autumn of 1944; in some cases, however, the patients were not available for re-examination before 1945, and in a few cases not till the beginning of 1946. Thus, of the original 689 cases (see table 2) 6 could not be followed up, and 683 cases therefore remained. Of these, 127 persons died during the course of the years, 16 of them of a disease, or as the result of accidents, having no direct connection with their rheumatic complaint. Five hundred and fifty-six cases were therefore left as a material for study, and to these the aforementioned questionnaire was sent out. Replies were received — in some instances only after repeated requests, however — from 544 persons (97.8 per cent). Of the remaining 12 persons with whom no contact whatsoever was made, 7 are women and 5 men who, according to the parish registers, are all

Table 2.

Showing whole series of cases grouped according to initial manifestation and to success of follow-up investigation.

Nature of initial manifestation	Whole series	No. of patients submitted to follow-up, incl. those who have died	No. of patients only replying to question-naire	No. of patients with whom no contact could be made
Arthritic symptoms[1]	521	498	12	11
Chorea[1]	127	118	4	5
Carditis alone . . .	41	39	—	2
Total	689	655	16	18
		(95.1 %)	(2.3 %)	(2.6 %)

living. Of the 5 men, information has been obtained regarding 1 of them from the military authorities and from other sources (treated at Sahlgren's Hospital, Gothenburg, in 1943 for a recurrence of rheumatic fever; signs of mitral insufficiency when discharged). As regards one of the 7 women, she was examined as late as 1941 at the out-patient department of the Children's Hospital, and then showed signs of a possible valvular lesion.

Of the 544 persons who replied to the questionnaire, 528 (95.0 per cent of the total 556 cases) could be re-examined. (Of these, 5 cases were not examined by me, personally, however, as the persons in question were living so far away that it was impossible for me to visit them; recent, liable information regarding their present state of health, in particular the condition of the heart, was, however, obtained from hospital records and from the record kept by their family physician.) Of the 16 persons who replied to the questionnaire but were not re-examined by me, personally, because of a refusal or for some other reason, 11 were women and 5 men. As regards 4 of the men, information was obtained regarding the medical examination carried out in connection with

[1] With or without carditis. (In the following tables this condition is always understood when these symptoms are mentioned.)

their compulsory military training or their service with the defense forces; in all 4 cases their military registration card contained the statement: "Heart, nothing of interest", or "Heart, no abnormality", or something else to the same effect. Although the information obtained from the military authorities is not, perhaps, fully reliable in all respects, these cases have nevertheless been included in some of the groups under consideration in my investigation.

As regards the patients who died, fairly complete and reliable information as to their state of health before death was obtained in every instance. Thus, if the 528 persons examined at the follow-up investigation are included, the course of life after the contraction of rheumatic fever was ascertained in 655 cases (= 96.0 per cent of 683 cases, or 95.1 per cent of the total series of 689 cases). If the 5 men about whom information was received from the military medical authorities are included, the corresponding figures will be 660 cases (96.6 and 95.8 per cent, respectively). If the remaining 12 persons who replied to the questionnaire but were not re-examined are also added, we have 672 patients whose course of life was more or less known; in other words, 98.4 per cent of the 683 patients with whom it was possible to make contact.

In connection with the follow-up investigation, the answers to the questions in the questionnaire were examined and checked, and where necessary they were supplemented. This measure, of first sending the patient a questionnaire to be filled in, was found to be more satisfactory than calling the patient up for an examination and then, in connection with this, asking him to answer the questions personally. Many of the patients, in fact, expressed their satisfaction at being able to think over the questions quietly and decide on the most suitable answers at home; they were thus able to question their parents on unclear points, and this was found especially valuable in cases where a long period had elapsed between the onset of the infection and the follow-up examination or when the patient in question had been fairly young at the time of the initial attack. The information thus obtained may therefore be said to be reliable.

Methods.

The patients who were available for re-examination were submitted to a general physical examination, at which special attention was paid to the condition of the heart and the signs obtainable at this examination which might indicate the presence of vitium cordis. In every patient, also, particular attention was paid to the condition of the joints at the time of examination. In addition, an electrocardiogram was made and a roentgenographic examination of the heart carried out in every instance. It was originally planned to make a phonocardiogram (by Mannheimer's method, 1940) also, with a view to obtaining a more objective recording of heart murmurs, but this plan had to be abandoned, unfortunately, owing to the impossibility of procuring an apparatus capable of fulfilling the requirements that would be placed on it. In order to control as far as possible that the patient had no active rheumatic disease a test for hemoglobin (Autenrieth) was carried out at the follow-up, and the microsedimentation rate of the red blood corpuscles was determined (by Landau's method). These two tests yielded normal values in practically every instance. (A few abnormally high microsedimentation rates were to be explained by the fact that the patient in question had just had, or still had, an acute upper respiratory tract infection or some other similar infection, and in cases where the elevated rate could not be explained in this way, a later control test yielded normal values.)

In the diagnosis and classification of the heart affections the principles outlined by the New York Heart Association in "Nomenclature and criteria for diagnosis of diseases of the heart" (1939 and 1945) were followed.

The heart volume was determined by roentgenography, by the method worked out by Liljestrand, Lysholm, et al. (1939), as well as by Jonsell (1939). Owing to defective technical facilities, however, it was not possible to take anteroposterior and lateral views simultaneously; they had therefore to be taken separately, in two different exposures. The resulting source of error with respect to volume determination was, however, according to

the roentgenologist who helped me with this examination, so small that it could be left out of account for my purposes. The focal distance was 1.50 meters. For the calculation of the heart volume, the formula used by Jonsell (1939) was employed, viz.:

$$V = k \cdot l \cdot b \cdot d$$

where k is the constant (= 0.42), l and b the long and transverse diameters of the cardiac ellipsoid in the frontal view, and d the depth of the heart or the "sagittal" diameter of the heart measured on the sagittal view. This total volume was then reduced down to a volume per square meter of body surface area, the body surface area being computed from Du Bois' (1927) formula. As regards the heart volume per square meter of body surface area, the values used at Swedish hospitals as the normal upper limits — not exceeding 450—500 cc for males (according to Liljestrand, Lysholm, et al.) and 420—450 cc for females — were employed. Volumes exceeding these were interpreted as pathologic, and indicated by the expression "enlargement of the heart," due allowance being made, of course, in borderline cases, for possible changes in shape. In several cases, the heart volume was within the limits stated as normal, while the heart silhuette showed pathologic features; in such cases the roentgenographic heart change has been described as "roentgenographic changes but no enlargement of the heart".

The electrocardiograms were in every case taken with the patient in the recumbent position. The electrodes were applied by means of clips on the forearms and the lower part of the left leg. In the chest lead, the fourth electrode was placed in the CR_4 position, according to the principles applied by the American Heart Association and the Cardiac Society of Great Britain and Ireland (1938), i.e. with one electrode in the left mid-clavicular line or directly to the left of it, over the apex of the heart, the other on the right arm, and with the apparatus switched into position as for lead I. For the chest lead, a metal disc was used, attached to the desired position by means of a suction device. Both in the chest and the extremity leads a compress soaked in

soda solution was placed between the electrode and the skin. The electrocardiogram was recorded, as a rule, after the patient had first taken a deep inspiration, then breathed out and held his breath for a short time.

In the interpreting of the electrocardiograms, both those taken at the follow-up investigation and those made at an earlier date, the normal values stated by Mannheimer (1940, p. 154), and by Scherf and Boyd (1945, pp. 21—24) were used.

No particular test for the cardiac function was applied when examining the heart. This was mainly owing to the fact that we are hardly in possession of a sufficiently unequivocal, and at the same time simple, test which could be carried out without difficulty on a large number of patients during a short visit. In consideration of the extent and nature of this investigation, it is to be expected that sufficiently reliable information on the functional capacity of the heart, its reserve power, and so on, ought to be obtainable by questioning the patients regarding possible symptoms of insufficiency in connection with fairly normal exertion such as walking up stairs or up hills, bicycling, outdoor walking and so on, with due consideration paid, of course, to those patients likely to have been in special training for these forms of exertion. Therefore, in order to gain some idea of these circumstances, a number of questions of a relevant nature were included in the questionnaire, and these were supplemented and widened at the follow-up examination. The statements thus obtained were then used as the basis for the functional classification.

Classification.

In addition to the natural classification into 1) a main group of cases with chorea[1] as the initial manifestation, the material was also divided, for certain purposes, with respect to the nature of the initial affection, into 2) a group with arthritic symptoms[1], and 3) a group with carditis alone. In the arthritis group were

[1] With or without carditis. (In the remainder of this publication this condition is always understood, when the above symptoms are mentioned.)

placed all cases of rheumatic fever, where subjective or objective pains in the joints had been present at the beginning of the attack in question and had lasted for some time, no consideration being paid to whether there had been signs of carditis or not at any time during the course of the disease. In view of the difficulty often experienced, especially for parents and relatives, in evaluating joint manifestations objectively, and as many patients are not presented for a medical examination till some time after all signs from the joints have completely disappeared (if they ever existed), the best measure seemed to me to include in the group with arthritic symptoms all cases where joint manifestations, even if they had only been subjective, had been present at any time during the rheumatic attack.

In the carditis group, on the other hand, I only included cases where neither choreic nor joint manifestations had existed, but only undoubted signs and symptoms from the heart. If joint or choreic manifestations subsequently appeared during the attack in question these have been indicated as a later complication (*not* as a recurrence).

Objections may, of course, be raised against this method of classification especially with regard to the division into carditis and arthritic symptoms. But this system has only been followed in connection with the preliminary study of the material, after which the groups were considered together where this was found to be more advisable. As has already been pointed out, a carditis component forms part of the picture in practically every form of rheumatic fever (Scherf and Boyd, and others). There are, of course, many transition forms between cases with pronounced manifestations from the joints (with or without carditis) and those with carditic symptoms alone. Cases with transient and merely subjective symptoms from the joints which are sometimes observed, could obviously, with every justification, just as well be counted among the carditis cases, since this manifestation must be said to be the dominant feature of the disease picture. As this latter distinction may have some justification, and possesses some interest, I have picked out the cases with barely distinguishable joint symptoms from among the cases with

arthritic symptoms, and compared them with the carditis cases, to see if any prognostic difference could be established between these two groups. More will be said about this further on.

Observation time.

The time elapsing between the onset of the rheumatic infection and the follow-up investigation shows a fairly high degree of variation. In all cases, however, it was at least 4 years. In 15 patients who had their initial attack before 1922 the observation time was more than 22 years (4 cases 23 years, 6 cases 24 years, 2 cases 25 years, and 3 cases 26, 27, and 28 years, respectively). In all the others, the observation time was between 4 and 22 years. The average observation time (see table 3) for the whole material was 12.2 years — 16.8 years for those patients in whom the onset occurred in the years 1922—1931, and 7.4 years for those who had their initial attack in 1932—1940.

Table 3.

Average number of years from initial attack to follow-up investigation (with respect to the 544 patients replying to the questionnaire).

Years of onset	Males		Females		All cases	
	No. of cases	No. of years	No. of cases	No. of years	No. of cases	No. of years
1922—1932	115	17.0	134	16.6	249	16.8
1932—1940	139	7.0	141	7.8	280	7.4
Total	261	11.9	283	12.5	544	12.2

Statistical methods.

In the statistical calculations the following methods were employed.

1. The standard error of percentage, $\varepsilon_p = \sqrt{\frac{p\,(100-p)}{n}}$

where p = percentage and n = number of cases.

2. The difference, d, between two independent samples, p_1 and $p_2 = p_1 - p_2$. The standard error of the difference, $\varepsilon_d = \sqrt{\varepsilon_{p_1}^2 + \varepsilon_{p_2}^2}$. When $\frac{n\,p}{100}$ is less than 5, or more than $n - 5$, the standard error obtained with this formula must be regarded as in some measure uncertain, being more uncertain if n is small.

3. The χ^2-method (worked out by R. A. Fisher and described by Bonnier—Tedin, 1940) was used as a test for heterogeneity and for comparison of different distributions.

P denotes probability that different groups belong to the same population.

$P \gtrless 0.05$ (corresponding to 2 times the standard error) denotes that there is a probable difference between the groups.

$P \gtrless 0.01$ (corresponding to 2.5 times the standard error) denotes that the difference is very probable.

$P \gtrless 0.001$ (corresponding to 3 times the standard error) denotes that the difference is significant.

d. f. = degrees of freedom.

For statistical methods used for the calculations of recurrence rates see chapter 5.

CHAPTER 2.

Incidence of rheumatic fever from various viewpoints.

Age and sex incidence.

Of the original 689 cases, 336 were boys (48.8 per cent), and 353 girls (51.2 per cent). (The difference between boys and girls is not statistically probable.) Table 4 shows these cases grouped according to age and sex, as well as to the nature of the initial attack.

In 127 cases (18.4 per cent) chorea was the initial manifestation of the disease. Of these 127 cases, 39 (30.8 per cent) were boys and 88 (69.2 per cent) girls; thus the proportion of boys to girls was approximately 3 to 7. In the entire chorea series, 74 per cent of the patients had their initial attack between the ages of 7 and 12 years, the peak incidence being at 10 years. As regards the boys, this peak falls at an earlier age, between 7 and 9 years, while as regards the girls it seems to lie just at the 10 year level. The number of boys is too small, however, to allow of any definite inference being made from the series. In only one case, a girl, did chorea appear before the age of 4 years, and among the boys there was no case before 5 years of age.

Arthritic symptoms as the initial manifestation of rheumatic fever were present in 521 cases (75.6 per cent). In this arthritis group, as mentioned before, were placed all cases with signs or symptoms from the joints, even if these were inconsiderable or merely subjective pain without any objectively demonstrable lesions. In this group, the boys were in the majority, with 282 cases (54.1 per cent), whereas there were only 239 cases (45.9 per cent) of the female sex. The difference is statistically very

Table 4.

Age at onset with respect to different initial manifestations, and sex distribution.

Age in years	Chorea with or without carditis			Arthritic symptoms with or without carditis			Carditis alone			All manifestations		
	Males	Females	Both sexes	Males	Females	Both sexes	Males	Females	Both sexes	Males	Females	Both sexes
0— 1	—	—	—	—	—	—	—	—	—	—	—	—
1— 2	—	—	—	—	1	1	—	—	—	—	1	1
2— 3	—	1	1	6	5	11	—	—	—	6	6	12
3— 4	—	—	—	4	7	11	2	1	3	6	8	14
4— 5	—	2	2	9	9	18	—	—	—	9	11	20
5— 6	4	5	9	16	10	26	2	—	2	22	15	37
6— 7	3	5	8	25	19	44	1	1	2	29	25	54
7— 8	7	7	14	30	22	52	2	4	6	39	33	72
8— 9	5	8	13	35	23	58	3	3	6	43	34	77
9—10	7	10	17	37	27	64	1	4	5	45	41	86
10—11	2	17	19	26	26	52	2	2	4	30	45	75
11—12	4	10	14	25	29	54	—	4	4	29	43	72
12—13	4	13	17	17	16	33	2	3	5	23	32	55
13—14	2	6	8	22	23	45	—	2	2	24	31	55
14—15	1	4	5	17	17	34	—	2	2	18	23	41
15—16	—	—	—	13	5	18	—	—	—	13	5	18
Total	39	88	127	282	239	521	15	26	41	336	353	689
Percentage of total number in the respective groups	30.7	69.3		54.1	45.9		36.6	63.4		48.8	51.2	
and its standard error	± 4.1			± 2.2			± 7.5			± 1.9		

probable. Here, a little over half of the cases, or 53.7 per cent, were among children between the ages of 7 and 11 years, inclusive, with a peak at 9 years. Here also, there seems to be a difference, though less marked, between boys and girls, the peak for the girls

falling at a slightly higher age (see table 4). In only one case was the patient under 2 years of age, more exactly, 18 months old, but as early as at 2 and 3 years of age the incidence of the disease begins to rise.

The remaining patients who had no signs or symptoms whatsoever from the joints were characterized as cases of carditis — 41 cases in all (5.95 per cent). Among these, the frequency curve begins to rise at 7 years of age, but after this age it is fairly even, without any particularly noticeable peaks up to the age of 13, when, as in the case of the other symptoms, it shows a strong drop. Among the carditis cases, the girls are in the majority. The difference is statistically very probable. In this group no cases occurred before 3 years of age.

The frequency curve relating to all cases of rheumatic fever (see table 4) shows a fairly smooth rise from 1 year of age up to and including 9 years, after which it sinks again, showing approximately the same incline as on the up side. In this series also, there is a definite lag in the girls' frequency curve as compared with that of the boys; among the boys the largest number of cases occurs at 9 years of age and among the girls at 10 years. The more striking increase in incidence, which seems to begin at 5 years among the boys, also starts one year later among the girls.

In order to gain some idea of the frequency with which the initial attack of rheumatic fever occurs after the age of childhood, and also of the frequency of the disease in different age-groups after the age of 16, I examined all the case records for the years 1922 to 1942, from all the Gothenburg hospitals, in which the diagnosis had been rheumatic fever, chorea, carditis, or corresponding synonyms. From the statements in these records, I endeavoured to ascertain as exactly as possible when the initial attack had occurred in each case. Especially in the case of an initial attack in early childhood, with recurrences at a more adult age, it must, of course, be borne in mind that the person in question may have forgotten not only *when* he was taken ill with similar symptoms previously, but also that he had been ill *at all* with such symptoms, and the chance of this forgetfulness occurring is probably even greater if the person in question was

Table 5.

Age at onset with respect to all cases where the initial attack occurred in the years 1922—1941 and which were treated at Gothenburg hospitals in the years 1922—1942.

Age at last birthday	Males	Females	Total	Age	Males	Females	Total	Age	Males	Females	Total
0	—	—	—	28	15	19	34	56	2	2	4
1	1	1	2	29	12	22	34	57	2	—	2
2	6	5	11	30	16	16	32	58	—	4	4
3	7	9	16	31	15	29	44	59	3	3	6
4	10	12	22	32	21	22	43	60	4	4	8
5	22	15	37	33	8	23	31	61	1	1	2
6	30	26	56	34	8	9	17	62	2	4	6
7	41	34	75	35	11	12	23	63	2	1	3
8	46	38	84	36	8	17	25	64	1	2	3
9	45	44	89	37	12	13	25	65	1	2	3
10	34	48	82	38	8	7	15	66	1	—	1
11	30	43	73	39	11	11	22	67	1	—	1
12	24	35	59	40	11	15	26	68	2	1	3
13	30	37	67	41	10	10	20	69	2	2	4
14	26	35	61	42	7	7	14	70	1	2	3
15	30	22	52	43	4	7	11	71	1	2	3
16	15	28	43	44	6	7	13	72	1	3	4
17	18	27	45	45	7	5	12	73	—	1	1
18	20	29	49	46	2	9	11	74	—	—	—
19	21	42	63	47	13	2	15	75	—	1	1
20	23	34	57	48	6	6	12	76	—	1	1
21	17	33	50	49	6	8	14	77	—	—	—
22	17	31	48	50	8	13	21	78	—	2	2
23	21	30	51	51	4	2	6	79	—	1	1
24	12	30	42	52	4	6	10	80	—	—	—
25	20	29	49	53	3	4	7	Total	872	1,119	1,991
26	21	32	53	54	2	3	5				
27	18	23	41	55	2	4	6				

	Males	Females	Total
1st quartile	218 (10 years)	280 (12 years)	498 (11 years)
Median	436 (19 »)	559 (20 »)	995 (20 »)
3rd quartile	654 (31 »)	839 (31 »)	1,493 (31 »)

Table 6.

Number of patients who had had the initial attack before a certain age.

	Number of cases		
	Males	Females	Both sexes
Before 16 years . . .	382	404	786
	(43.8 % ± 1.7)	(36.1 % ± 1.4)	(39.5 % ± 1.1)
Before 20 years . . .	456	530	986
	(52.3 % ± 1.7)	(47.4 % ± 1.5)	(49.5 % ± 1.1)
Before 25 years . . .	546	688	1,234
	(62.6 % ± 1.6)	(61.5 % ± 1.5)	(62.0 % ± 1.1)
Before 30 years . . .	632	803	1,445
	(72.4 % ± 1.5)	(71.8 % ± 1.4)	(72.6 % ± 1.0)
Before 50 years . . .	822	1,038	1,870
	(94.3 % ± 0.8)	(92.8 % ± 0.8)	(93.4 % ± 0.6)
Total no.	872	1,119	1,991

treated at home for the initial attack and if the symptoms of, for instance, the joint affection, were slight or only moderate. (As regards chorea and carditis the same comments can be said to apply here as those previously put forward in connection with children — see p. 52.)

Consequently, one must count on such a frequency curve, on account of incorrect statements as to age at the initial attack, being, if anything, too much displaced in the direction of higher ages rather than the opposite. The incompleteness of the case records, and the lack of uniformity between the diagnoses of various physicians, often made it difficult to pick out the cases with true rheumatic fever from among records with so many different diagnoses ("polyarthritis acuta," "p. subacuta," "p. subchronica," "p. chronica," "p. recidivans," and so on), but those cases that I have accepted for this investigation may on the whole be regarded as cases of genuine rheumatic fever. With these reservations, the series assembled would seem to present a fairly acceptable picture of the occurrence of rheumatic fever, for the

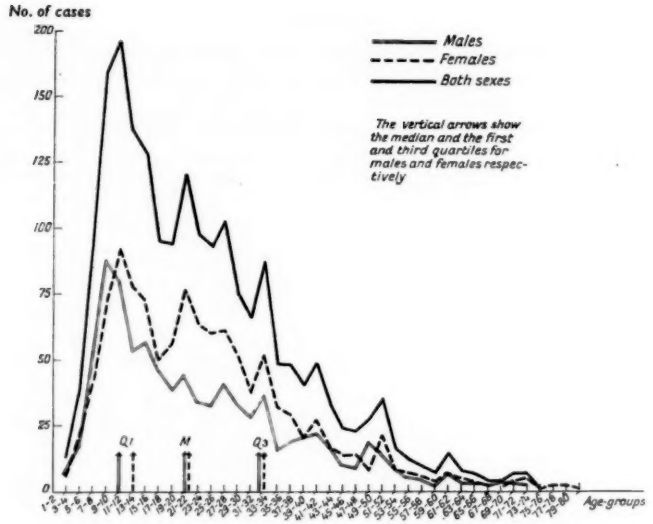


Fig. 1. Age at onset with respect to all cases of rheumatic fever (incl. chorea) where the initial attack occurred between 1922 and 1941 and which were treated at one of the Gothenburg hospitals. (The cases have been assembled into age-groups of 2 years each.)

years following those of childhood, in the city of Gothenburg during this limited period. Only patients who, according to the case records, had had their initial attack in one of the years between 1922 and 1941, inclusive, were included in my series. These cases, as well as similar cases from the Children's Hospital for the corresponding years[1] are assembled in table 5, and in addition to this a frequency curve relative to this table was constructed after the cases had been grouped together into age groups of 2 years (fig. 1). With the above-mentioned reservation, it is quite evident from this curve that the peak incidence for the onset of rheumatic fever, during this 20 year period in the city of Gothenburg, occurs, in fact, in the years of childhood, at 9 years of age, and that after this age the curve falls away fairly rapidly until,

[1] In order to obtain all cases for a 20 year period, patients treated at the Children's Hospital during 1941 were also included.

at about 45—50 years of age it is very low and begins to assume a more level appearance. The previously mentioned possibility, that some of the adults might have forgotten, or even perhaps have never been aware, that they had had a mild initial attack as a child, would only cause the curve to show an even more pronounced peak in the early years of life and a more rapid drop in later years.

Table 6 shows calculations made from table 5, proving that nearly two-thirds, or 39.5 per cent, of all initial attacks occur before the age of 16, nearly one-half, or 49.5 per cent, before 20 years of age, and nearly three-fourths, or 72.6 per cent, before the age of 30. After 50 years of age, the incidence of initial attacks seems to be low, 6.6 per cent; this figure, in view of what has already been said, may even be said to be on the high side.

In other words, rheumatic fever is a disease arising in the vast majority of cases in the years of childhood and early life, a circumstance that it would seem necessary to stress.

Looking at curve 1 and table 5, from the standpoint of the relation between the sexes, it will be seen that, besides the lag (previously observed in the child material also) in the curve relative to the females as compared with that of the males, the number of female cases seems to be considerably larger than the number of males, except in the very earliest years of childhood. The difference between them is also statistically significant.

If one picks out the cases with chorea as the initial manifestation from among the above-mentioned total series of cases with an initial attack of rheumatic fever in Gothenburg during a 20 year period, one will find that chorea as a primary manifestation of rheumatic fever practically never occurs after the age of 20 nor before the age of 4. Only 1 case occurred before the age of 4 years and 1 after the age of 20 (fig. 2). This finding is in good agreement with the experiences in other quarters (Holland Clarke, 1940; Hedley, 1940; Wedman and Wedman, 1944). It is clear from the histogram that the majority of the cases with chorea as the initial manifestation occur before the age of 16, and only in 7 per cent of this material was this lesion the primary evidence of the disease in patients over the age of 16. This survey

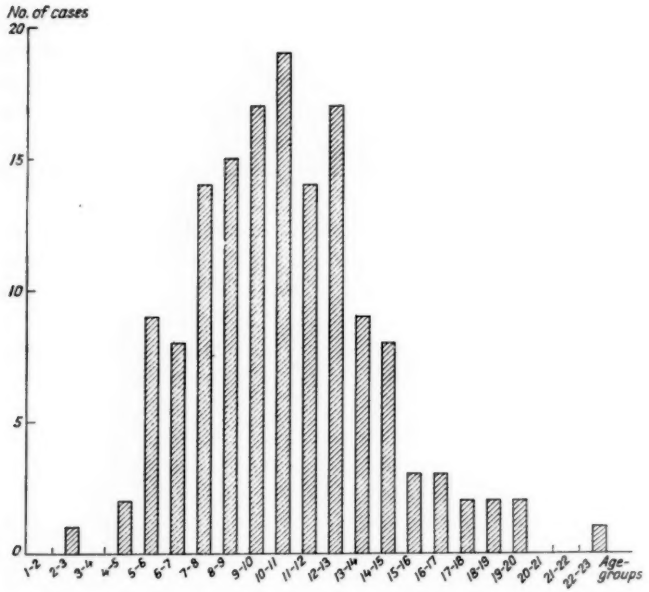


Fig. 2. Age at onset with respect to all cases where the initial manifestation of rheumatic fever was chorea. (Total material from all hospitals in Gothenburg.)

of all cases with chorea as the primary lesion treated at the hospitals in Gothenburg brought out no essential alterations in previously mentioned figures relative to the relation between the sexes, the proportion of males to females being 29.5 per cent: 70.5 per cent, in other words as 3:7.

Annual and seasonal variations.

As in the case of so many other diseases, so also in rheumatic fever, certain variations in its seasonal as well as in its annual incidence are to be expected.

Table 7 shows a survey of the whole series of cases worked up for the purpose of illustrating variations in the incidence of onset between different years, and on the basis of this table the histo-

Table 7.

Showing the initial attack graded according to the calendar year at onset and the nature of the attack in the whole series of cases.

The table gives the number of cases in each group. Figures within brackets denote patients who had the initial attack during the year indicated in the table but were not admitted to the Children's Hospital till 1941—1942.

Calendar year at onset	Chorea	Arthritic symptoms	Carditis alone	Total cases
Prior to 1922 . .	11	17	2	30
1922 . .	8	17	4	29
1923 . .	6	20	—	26
1924 . .	6	13	3	22
1925 . .	8	15	1	24
1926 . .	3	37	1	41
1927 . .	12	30	—	42
1928 . .	8	32	4	44
1929 . .	5	38	3	46
1930 . .	12	32	7	51
1931 . .	3	20	—	23
1932 . .	12	20	—	32
1933 . .	6	24	2	32
1934 . .	6	34	1	41
1935 . .	2	16	1	19
1936 . .	4	17	2	23
1937 . .	2	22	2	26
1938 . .	6	37 (+ 1)	2	45 (+ 1)
1939 . .	5 (+ 1)	55	4	64 (+ 1)
1940 . .	2	25 (+ 1)	2 (+ 1)	29 (+ 2)
Total	127 (+ 1)	521 (+ 2)	41 (+ 1)	689 (+ 4)
1941 . .	1	9	3	13
1942 . .	1	18	1	20
			Total	726 cases

gram in figure 3 was constructed. For the sake of comparison, and since rheumatic fever, as will be demonstrated later on, shows a certain predilection for the winter months, the material was also grouped by adding the last half-year of each year to the first half-year of the following year. In this way, a histogram (fig. 4) was obtained, with every column including one whole winter season. In both figures, also, the patients contracting the disease in the years 1941 and 1942 (or treated at the hospital for the first time in those years) were added in order to demonstrate that the strong increase in incidence during the years 1938—1940 was followed by an equally strong drop during the years 1941—1942.

This table, as well as the two histograms, demonstrate clearly that there was a fairly pronounced variation in yearly incidence in my material. Thus, there was a noticeable decrease in the number of initial attacks during the season 1923—1924, then a gradual increase during the next few years up to 1929—1930, after which the incidence dropped again fairly rapidly to a minimum in 1931—1932. From this date, the number of initial attacks began to increase again, reaching a maximum, although not so marked as the previous one, during the season 1933—1934. The decrease subsequently ensuing reached its lowest level during the season 1935—1936, remaining at that level during the following season 1936—1937; then there is another increase to an absolute maximum in the season 1938—1939, the incidence being highest during the year 1939. After this, there was again a decrease in the number of initial attacks, which reached a minimum during the two seasons 1940—1941 and 1941—1942, where the absolute minimum occurred in the year 1941. Because of the fairly small number of chorea cases, as well as of those with carditis alone (see table 2), the variations relative to these cases were not so striking, and therefore not suitable as material for special attention.

A similar histogram showing the distribution of the recurrent attacks (fig. 5) on the different years shows, it is true, certain variations, which to a certain extent, also, seem to coincide with the variations in the initial attacks, but this feature is less marked, a fact which probably can be partly explained by the small number

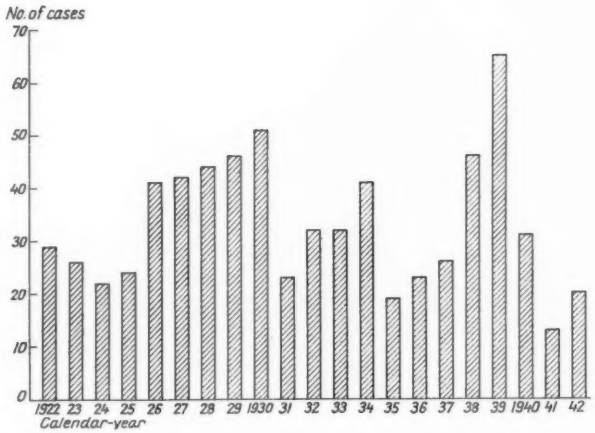


Fig. 3. Calendar year of initial attack with respect to all cases of rheumatic fever (incl. chorea) treated at the Children's Hospital in Gothenburg during the years 1922—1942.

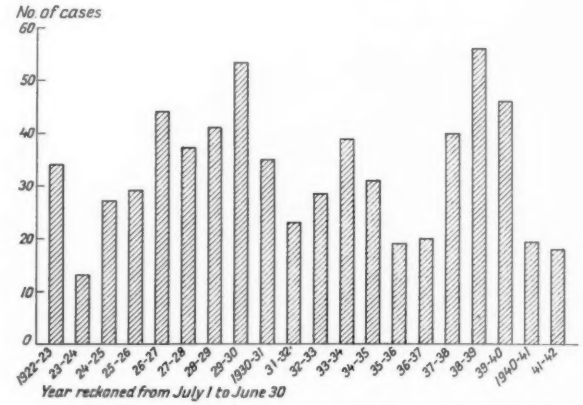


Fig. 4. Same material as in Fig. 3, but using a year reckoned from July 1 to June 30. [64 cases in which either the season of onset was unknown (treated for initial attack at home) or the initial attack occurred during the first half of 1922 or the second half of 1942 are not included in the histogram.]

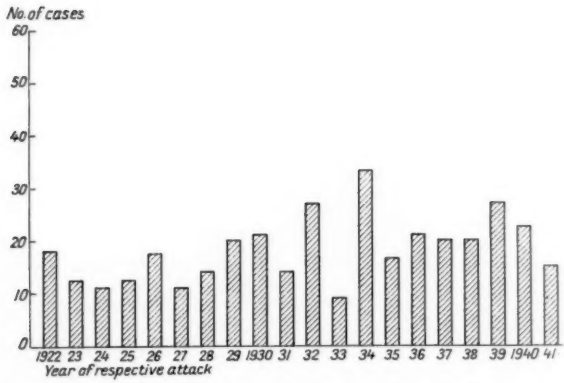


Fig. 5. Number of recurrent attacks classified according to the year of onset of the attacks (in all, 360 attacks).

Table 8.

Showing the initial attack graded according to the month of onset in the 631 cases where the exact month could be established. The table gives the number of cases in each group.

Month of onset	Chorea	Arthritic symptoms	Carditis alone	No. of males	No. of females	Total cases
January .	13	40	6	26	33	59
February .	7	29	5	19	22	41
March . .	16	32	2	22	28	50
April . .	11	32	2	23	22	45
May . . .	6	50	4	32	28	60
June . . .	4	47	2	27	26	53
July . . .	6	40	1	22	25	47
August . .	9	31	2	20	22	42
September	11	33	3	25	22	47
October .	8	40	3	29	22	51
November	9	53	6	32	36	68
December .	12	51	5	27	41	68
Total	112	478	41	304	327	631

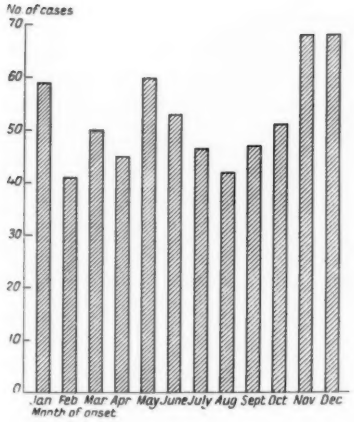


Fig. 6. Initial attack classified according to month of onset in the 631 cases (out of a total of 689 cases) where this month was known.

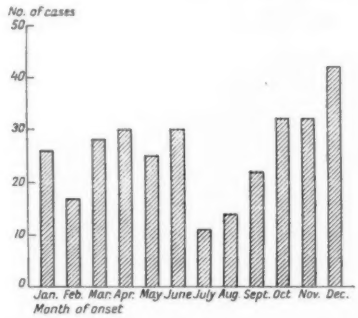


Fig. 7. Total recurrent attacks classified according to month of onset in the 309 recurrences (out of a total of 418 recurrent attacks in the whole material) in which this month was known.

of recurrent attacks where the month of onset was known (360) occurring in the period under discussion.

As regards seasonal variations, the findings are shown in table 8 and in the histogram in figure 6. Although the number of cases is fairly evenly distributed over the whole year there are nevertheless two definite minimum levels, one during the months

February—April, and the other during the months July—September, with an average of 45.3 initial attacks a month in each. As regards the two maximum levels, the May—June period shows an average number of 56.5 cases a month, while the more pronounced maximum, occurring during the months November—January, shows an average of 65.0 cases a month. If we compare the average monthly incidence of the disease in these three winter months, November to January, with the same incidence in the other months of the year, we find that the average monthly incidence for February to October is 48.4 cases. It thus ought to be fairly evident that the incidence of rheumatic fever shows at any rate a maximum during the months November—January.

Similar conditions, although less striking (due to a smaller series of cases), are found with regard to the distribution of the recurrent attacks (fig. 7). The most noticeable feature here seems to be the remarkably small number of recurrent attacks during the summer months July—August (—September).

As regards the reason for these annual and seasonal variations, it is my intention to take up this question for closer analysis in a later report, making particular reference to the dependence of the variations on various weather factors and to the possible connection between rheumatic fever and other diseases, particularly scarlatina.

CHAPTER 3.

Has the material changed character in any way during the course of time? Can any change in the prognosis be detected for later years?

In a paper dealing with "Juvenile Rheumatism" in London, Francis Bach et al. (1939) made the following statement: "It can therefore be asserted, with a certain degree of confidence, that even if there be no notable decrease in the incidence of rheumatic infection, there has been a definite decrease both in the virulence of the acute form of the disease and in the frequency and severity of the cardiac complications". Karlström (1940), also, stated in a report on juvenile chorea minor and acute polyarthritis in Stockholm for the 20 year period 1920—1939 that the number of children suffering from chorea minor showed a strong decrease both as regards absolute numbers and in relation to the population figures, and that the disease had become much less severe during this period. On the other hand, he could observe no similar features with respect to acute polyarthritis, which showed no alterations either with regard to frequency or to character.

Is there anything in the present investigation to indicate similar changes? Is the material heterogeneous in any way? In order to obtain an answer to these problems I intend first to study the conditions with respect to chorea minor during the period covered by my investigation.

As may be seen from table 9, a slight decrease seems to have occurred in the number of initial attacks of chorea minor during the nineteen-thirties, a feature even more apparent if the cases are grouped into 4-year groups. In figure 8, is shown a histogram which provides an even better illustration of the decrease

Table 9.

Showing the cases with chorea as the initial manifestation of rheumatic fever and the total cases with an initial attack of rheumatic fever (incl. chorea) classified according to the calendar year at onset.

The figures within brackets denote cases admitted to the hospital for the first time subsequent to the year 1940. — Relation between number of cases with chorea and total number of cases treated at the medical clinic of the hospital during the different year-periods tested with the χ^2-method for heterogeneity; relation between number of cases with chorea (as the initial manifestation of rheumatic fever) and total number of cases with an initial attack of rheumatic fever tested in the same way.

Age at onset	No. of cases with chorea		No. of cases treated at med. clin. of hospital		Percentage of cases of chorea (init. attack) among cases treated	Total no. of cases with initial attack of rheumatic fever		Percentage of cases with chorea in the total no. of cases with rheumatic fever	Total no. of children between the ages of 3 and 15 in Gothenburg
Prior to 1922	11								
1922	8		1,106			29			
1923	6	28	1,140	4,637	6.04	26	101	27.8	45,717
1924	6		1,238			22			(year 1920)
1925	8		1,153			24			
1926	2		1,134			41			
1927	11	25	1,144	4,616	5.41	42	173	14.5	
1928	7		1,206			44			
1929	5		1,132			46			
1930	12		1,209			51			
1931	3	32	1,148	4,700	6.81	23	138	23.2	47,494
1932	12		1,133			32			(year 1930)
1933	5		1,210			32			
1934	6		1,203			41			
1935	2	13	1,186	4,857	2.68	19	109	11.8	44,776
1936	3		1,172			23			(year 1935)
1937	2		1,296			26			
1938	6		1,205			45 (+ 1)			
1939	5 (+ 1)	15	1,439	5,491	2.55	64 (+ 1)	142	10.5	41,174
1940	2		1,234			29 (+ 2)			(year 1940)
(1941)	(1)		(1,613)			(13)			
					$\chi^2 = 15.7$, $P < 0.01$, d. f. 4			$\chi^2 = 18.9$, $P < 0.001$, d. f. 4	

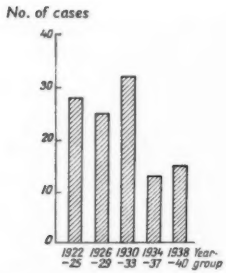


Fig. 8. No. of patients with chorea as the initial manifestation of rheumatic fever divided into year-groups according to the year of onset.

in the number of chorea cases. The decrease in incidence seems, in reality, to occur subsequent to the years 1933—1934. If these chorea cases be placed in relation to the total number of rheumatic fever cases in the respective year groups a fairly continuous decrease during the course of the years will be found, this proving to be statistically significant when tested by the χ^2-method for heterogeneity.

Unfortunately, I was not able to compare the number of chorea cases with the total number of children of corresponding ages in Gothenburg, as no such statistical figures are available with regard to Gothenburg, with the exception of a few definite census years. An attempt to obtain these figures would have involved almost insuperable difficulties. Table 9, however, includes the available figures for the number of children in Gothenburg during these special census years, and from these it would appear that no particularly noticeable changes in the number of children have taken place. The drop in the number of children from 1920 to 1940 shown in this survey only amounts to about 12 per cent.[1]

If, on the other hand, we compare the chorea cases with the number of children treated at the Medical Department of the Children's Hospital during these years, the same decreasing

[1] It should be pointed out that among the children in this material which were treated at the Children's Hospital some were not living in the City of Gothenburg. There were so few of them, however, that their number can be of no significance in the present connection.

tendency will be observed; this is found by χ^2-analysis to be statistically very probable. The temporary, both absolute and relative, increase in the number of cases in the years 1930—1933 is, however, remarkable.

As may be seen from table 9, no corresponding decrease could be found in the numbers of the other cases of rheumatic fever. The fluctuations in number, which appear to occur among these other cases, are probably to be explained by the previously mentioned periodical rise and fall in the incidence of the disease.

In order to form an idea of whether the chorea had altered as regards the degree of severity during this period, all cases with chorea at the initial attack were divided up (in the main according to the same method as that adopted by Wilson, 1940) into cases with slight, moderate, or severe chorea.[1] This method yielded nothing to indicate that the chorea had become milder as time passed; if anything, the figures obtained pointed in the opposite direction, although the variations observed can probably be put down to chance. The comparatively small number of chorea cases, also, allows of no definite inferences being made regarding possible changes in the recurrence rate or in the risk for a fatal outcome in later attacks with the same or other forms of rheumatic fever. Nor does the degree of the cardiac affection give any guidance on this point, since in practically all cases the cardiac affection was described as not definitely established or slight.[2] It should perhaps be mentioned, however, that during

---

[1] The chorea was considered of *slight* severity if the choreiform movements were mild, and of less than one month's duration. It was considered of *moderate* severity if the movements were of moderate intensity and of less than one month's duration, or if the movements were of slight intensity but of more than one month's duration. Finally, the chorea was regarded as *severe* if the choreiform movements were extreme, no consideration being paid to their duration, or if they were of a more moderate nature but of more than one month's duration.

[2] The cardiac affection was considered to be *not definitely established* in cases where no definite signs of active carditis could be detected during the active infection either at the physical examination or by an electrocardiographic or roentgenographic examination. In cases with carditis, this was interpreted (using approximately the same method of classification as that

the years 1922—1931 there were 3 cases with moderate and 3 cases with severe cardiac affection in connection with the initial attack, while during the years 1932—1940 only one case with severe cardiac affection at the initial attack was encountered. As regards patients who died sooner or later, the two mentioned year-groups yielded 6 (8.8 per cent) and 3 (7.0 per cent) cases, respectively.

Passing now to the other two groups, cases with arthritic symptoms with or without carditis and cases with signs of carditis alone, a classification according to the severity of the joint affection is found to be of little value from this point of view, and is, besides, fairly difficult to carry out satisfactorily. Furthermore, according to all the experience gained by well-known clinicians, it is the heart and the degree of cardiac affection alone that will determine the course and severity of the rheumatic affection. Consequently, these two groups (cases with arthritic symptoms and cases with carditis alone) have been clubbed together in the present connection, and consideration has only been paid to the severity of the cardiac affection. This was classified, when studying the individual case records, as "not definitely established," "slight," "moderate," and "severe" carditis. As the individual groups obtained by this system were too small to be of much value for purposes of comparison, cases with not definitely established and mild cardiac affection were classed as one group and a similar measure was adopted with the moderate and severe cardiac affections.

---

used by Wilson, 1940) as *slight* when the signs of cardiac affection were of slight intensity (tachycardia, valvulitis, mild myocarditis with retardation in the P—Q time or other electrocardiographic changes of an inconsiderable and transient nature) and of less than one month's duration. The carditis was said to be of *moderate* severity in cases where more pronounced signs of carditis without congestive failure were present (pericarditis, moderate dilatation, fairly severe heart block with dropped beats or complete heart block, precordial pain, and so on), or mild, subacute symptoms of more than one month's duration. Among cases with *severe* carditis were placed those presenting signs of congestive failure or those with signs of moderate severity lasting longer than one month.

Table 10.

Frequency of »severer cases» during different years.

The table does not include 47 cases treated at home during the first attack nor 19 cases in which the initial attack occurred before 1922.

Calendar year at onset	Total cases contracting the disease		No. of cases with »moderate» or »severe» carditis[1]		Percentage of total
1922	16	60	8	27	45.0
1923	17		4		
1924	12		7		
1925	15		8		
1926	33	121	7	34	28.1
1927	20		4		
1928	34		11		
1929	34		12		
1930	35	96	11	37	38.5
1931	17		5		
1932	18		11		
1933	26		10		
1934	32	85	10	30	35.3
1935	16		5		
1936	17		7		
1937	20		8		
1938	38	121	10	32	26.4
1939	57		16		
1940	26		6		

Table 10 shows the material classified in accordance with this system. From this it would seem that while the total number of severe cases (i.e. those cases where the cardiac affection was of moderate or severe intensity) appears to remain about the same in the various years, the percentage incidence of severe cases shows a falling tendency, this decrease appearing to be concentrated chiefly to the period immediately following the years

[1] For classification see text.

1922—1925. During the following years it appears rather as if there were certain fluctuations in the percentage incidence of the severe cases without there actually being any definite tendency towards a drop. It should be pointed out that the reduction in the number of severe cases noted after the years 1922—1925 may possibly be explained by the fact that during the period 1922—1925 a larger number of patients with mild attacks may have been treated at home during the whole of their illness than was the case during later years. Because of this, the percentage incidence of moderate and severe cases in 1922—1925 is perhaps larger than it actually ought to be. It is possible that the increased confidence in the Children's Hospital and its physicians, as well as the increasing realization of the importance of early, consistent treatment of children suffering from this disease, has acted as an incentive during more recent years to bring in the children to the hospital to a greater extent than was previously the case, especially during the first years covered by this investigation.

In order to investigate in another manner, whether the material under discussion was heterogeneous in any way, I decided to study the frequency of pericarditis[1], as it seemed to me that the relative occurrence of such a well-marked complication as pericarditis must constitute a more objective basis for the evaluation of possible changes in the character of the disease than the previously mentioned classification into mild and severe cardiac lesions. Pericarditis is, it is true, not always diagnosed in the living, as was mentioned before, but in all probability it can be assumed that the proportion of cases not detected at the clinical examination is fairly even in the various years, and this aspect therefore need not be considered to have much significance in the present connection. It is, of course, possible, however, that with increasing experience the number of cases with clinically demonstrated pericarditis will increase

[1] Cases with chorea as the initial manifestation have not been included here, as the frequency of pericarditis at any time during the subsequent course was very low among these cases — 3 cases in the 1922—1931 year-group and 1 case in the 1932—1940 year-group.

Table 11.

Occurrence of clinical findings of pericarditis at the initial attack or a later one in relation to the total cases with an initial attack.

(The cases with chorea as the initial manifestation of rheumatic fever are not included here.)

Relation between occurrence of pericarditis and total cases of rheumatic fever during the five-year periods tested with the χ^2-method for heterogeneity.

Initial attack in year-group:	Total cases with rheumatic fever (including cases treated at home for initial attack)	No. of cases with pericarditis	Percentage incidence of pericarditis
1922—25 . . .	70	19	27.1
1926—29 . . .	138	27	19.6
1930—33 . . .	105	24	22.9
1934—37 . . .	93	17	18.3
1938—40 . . .	124	17	13.7
		$\chi^2 = 6.1$, $P < 0.20$, d. f. 4	

slightly as time passes. Finally, it should also be pointed out that when the cases were classified into groups with not definitely established, slight, moderate, or severe cardiac lesions, all the pericarditis cases were included in the last-mentioned two groups; the pericarditis group now to be discussed ought therefore to constitute a group of cases with cardiac disorder of moderate and severe intensity classified in a more objective manner.

Table 11 shows all the cases with clinically observed pericarditis, grouped according to the year of onset and distributed over four-year groups as in earlier tables. (Cases developing pericarditis at an attack subsequent to the initial episode are also included here.) As will be seen, the percentage incidence of pericarditis shows a tendency to drop gradually in all year-groups (but with a certain tendency to increase temporarily in the 1930—1933 year-group). A χ^2-test for heterogeneity, however, gives no grounds for assuming that chance alone might not have been responsible for this, since the existing heterogeneity

cannot be described as probable from the statistical standpoint. If we limit the comparison to the 1922—1925 and 1938—1940 year-groups we see, however, that there is a statistically probable difference between them.

It can, of course, be objected, that in the last year-group, at any rate, there are one or more patients who had not yet had their pericarditis, patients who, in other words, were destined to develop pericarditis in a later attack which they had not yet had. This risk must be said to be very small, however. As will be shown later on (see p. 166), not less than 70.0 per cent of the total series (82 out of 117 cases) developed their (clinically detected) pericarditis at the first attack, and 24.8 per cent (29 out of 117 cases) at the second attack. As, besides, in two-thirds of the cases in the total series (67 per cent), the second attack occurred before the end of the fourth year following the recovery from the first attack, and as all cases had been observed for at least 4 years, this objection may be said to have little significance. — It should furthermore be remembered that the higher frequency of pericarditis among the cases where the initial attack occurred between the years 1922 and 1925 may at least in part be due to the fact that a number of cases with mild signs and symptoms without further recurrence perhaps never came to the notice of the hospital.

The investigations reported up to the present thus provide certain grounds for the belief that the material under discussion in some respects is heterogeneous, although statistical proof to this effect was not obtained in all calculations. The figures mentioned indicate, however, that in recent years there has been a certain tendency in the direction of milder and less complicated cases. A study of the mortality yields the final proof that such is the case.

As regards the study of the mortality also, I considered it advisable not to include the patients who had had chorea at the initial attack, since, as will be mentioned later on (see p. 108), the mortality rate is so much lower in this group than it is for other cases. On the other hand, cases with arthritic symptoms

Table 12.

Deaths in the various groups in relation to the total rheumatic fever material (including cases treated at home during the entire initial attack).

Relation between cases dying at the initial attack and total number contracting the disease tested with the χ^2-method for heterogeneity in group A.

Initial attack in year-group:	No. of cases	No. of deaths at initial attack	%	Total deaths (at time of follow-up)	%
A. *Cases with arthritic symptoms at onset.*					
1922—25 . . .	63	11	17.5	20	31.7
1926—29 . . .	130	9	6.9	28	21.5
1930—33 . . .	96	10	10.4	16	16.7
1934—37 . . .	88	4	4.5	6	6.8
1938—40 . . .	116	4	3.4	5	4.3
		$\chi^2 = 13.7$, $P < 0.01$, d. f. 4		$\chi^2 = 33.0$, $P < 0.001$, d. f. 4	
B. *Cases with cardiac symptoms alone at onset.*					
1922—25 . . .	7	3	42.8	5	71.5
1926—29 . . .	8	5	62.5	7	82.5
1930—33 . . .	9	3	33.3	3	33.3
1934—37 . . .	5	1	20.0	2	40.0
1938—40 . . .	8	1	12.5	1	12.5

at the initial attack were grouped together with cases where the primary manifestation was carditis alone. The tendency is the same, in point of fact, in both these groups, as may be seen from table 12. As, furthermore, the advisability of dividing these into two groups can be considered dubious from several points of view, the method I have adopted would seem to be justifiable, and in any case it can have no appreciable effect on the conclusions drawn. (Because of the comparatively small number of cases of primary carditis in each year-group, the heterogeneity in this group cannot be tested by any suitable statistical method.)

As may be seen from table 13 and figure 9, there has been a definite drop in the primary mortality (by which I mean, in

Table 13.

Number of deaths at initial attack and total deaths (up to Jan. 1, 1945) in relation to total number of patients contracting rheumatic fever (including cases treated at home during initial attack).

The table refers to cases with arthritic symptoms or carditis alone at onset (not cases with chorea as the initial manifestation). Relation between total number of rheumatic fever cases and mortality during the five-year periods tested with the χ^2-method for heterogeneity.

Initial attack in year-group:	No. of cases	Deaths at initial attack	%	Total deaths	%
1922—25 . . .	70	14	20.0	25	35.7
1926—29 . . .	138	14	10.1	35	25.4
1930—33 . . .	105	13	12.4	19	18.1
1934—37 . . .	93	5	5.4	8	8.6
1938—40 . . .	124	5	4.0	6	4.8
		$\chi^2 = 16.0$, $P < 0.01$, d. f. 4		$\chi^2 = 40.8$, $P < 0.001$, d. f. 4	

this connection, the mortality occurring as the result of the first attack) during recent years, both as regards the total number of cases and the percentage incidence. A χ^2-test proves, also, that this decrease is statistically very probable. If one takes into consideration the possibility that the years 1922—1925 do not represent the total number of cases of rheumatic fever in the same way as the following years, as more cases with mild signs or symptoms may possibly have been treated at home in those years, one finds that the same tendency is present during the years 1926—1940, although here a χ^2-test does not reach the borderline of what is usually considered to be statistical probability. (For these four year-groups $\chi^2 = 6.5$, $0.05 < P < 0.10$, d.f. 3.)

Turning now to the total mortality (by which is meant here all patients who, up to the time of the follow-up, had died in direct connection with an attack (initial attack or recurrence) of rheumatic fever or as a direct consequence of the heart lesion

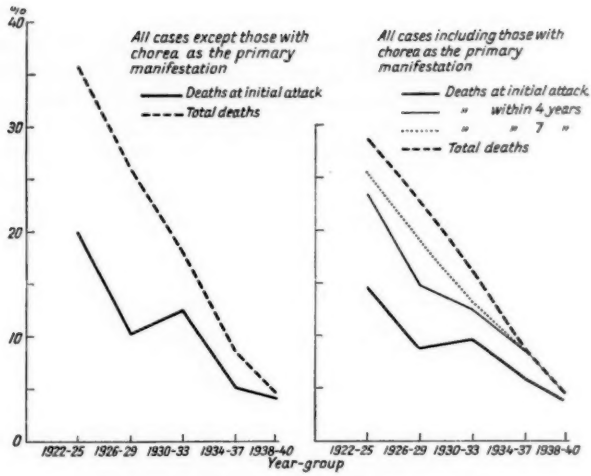


Fig. 9. Showing deaths in various year-groups.

produced by such an attack), analogous conditions are found (table 13, fig. 9). Here also, the probability of a successive decrease in the number of deaths is statistically significant. Excluding the 1922—1925 year-group, and making a χ^2-test on the remainder, we still find a statistically significant difference ($\chi^2 = 25.7$, $P < 0.001$, d.f. 3).

A comparison between the curves in figure 9 indicates that as regards the mortality from the initial attack there have been a number of fluctuations in the falling tendency during the course of the period under investigation, while the curve relative to the total mortality shows a smooth and undoubted decrease in the percentage incidence. It is obvious, here, that time is acting as a levelling factor.

The objection can naturally be raised here, that while, in the study of the total mortality, the percentage figures correspond on the whole to the death rate in the respective year-groups, these figures are not directly comparable with one another, since the more recent cases in the material have not yet been under observation for as long a time as the earlier cases. In consequence

Table 14.

Mortality in the whole material (including cases with chorea as the initial manifestation).

Relation between total number of rheumatic fever cases and mortality during the five-year periods tested with the χ^2-method for heterogeneity.

Initial attack in year-group:	Total cases	Deaths at initial attack		Deaths within 4 years		Deaths within 7 years		Total deaths	
			%		%		%		%
1922—25 .	98	14	14.3±3.5	23	23.4±4.3	25	25.5±4.4	28	28.6±4.6
1926—29 .	163	14	8.7±2.2	24	14.7±2.8	31	19.0±3.1	37	22.7±3.3
1930—33 .	137	13	9.5±2.5	17	12.4±2.8	18	13.1±2.9	22	16.1±3.1
1934—37 .	106	6	5.7±2.3	9	8.5±2.7	9	8.5±2.7	9	8.5±2.7
1938—40 .	137	5	3.7±1.6	6	4.4±1.8	—	—	6	4.4±1.8
Total	641 [1]								
		$\chi^2 = 9.9$, P < 0.05, d. f. 4		$\chi^2 = 21.6$, P < 0.001, d. f. 4		$\chi^2 = 12.6$, P < 0.01, d. f. 3			

of this, the total number of deaths in the first half of the material must be larger than the corresponding number in the latter half. However, if we calculate in each group the death rate for a 4-year period following the onset, we obtain, nevertheless, a statistically significant decrease in the number of deaths.

For the sake of completeness, I have assembled in table 14 the death rates for all cases (inclusive of the chorea cases), firstly in connection with the initial attack, and secondly after the passage of 4 and 7 years, respectively, from the onset of the initial attack. In figure 9 curves have been made to illustrate these death rates. As may be seen from table 14, the mortality within 4 years of the initial attack shows a statistically significant drop during the observed period 1922—1940. The mortality rate for the first 7 years after the onset shows the same features, the drop in the number of deaths being statistically very probable.

[1] 30 cases with initial attack before 1922 and 18 cases not re-examined are not included in the total figure.

It seems to me to be of interest in this connection to investigate whether the recurrence rate also has altered in any way during the course of the years. Here also, I did not include the cases with chorea as the initial manifestation of rheumatic fever in my study, partly because the chorea cases (as will be shown later) seem to show a somewhat stronger tendency to recurrence during the first years after the initial attack; as, furthermore, the number of cases with chorea as the primary manifestation so strikingly decreased towards the end of the period studied, to include these cases might conceivably have yielded a slightly skew picture of the situation. (An inclusion of these cases with primary chorea causes, however, no essential change in the conditions demonstrated here.)

A study of table 15 would seem to indicate that the tendency to a first recurrence is greatest in the 1922—1925 year-group while the following year-groups seem on the whole to show the same recurrence rate. [The difference between the 1922—1925 year-group and the following three year-groups counted together, is statistically significant when calculated on 7 years (22.4 % $\pm$ 7.1).] This may conceivably be due in part to the fact that more cases with the initial attack in the years 1922—1925 had been treated at home during the whole of this attack and then had had no recurrent attacks, thus not coming up for observation at the Children's Hospital.

If we divide the material approximately into two halves (year-groups 1922—1931 and 1932—1940), by which measure the second half will include only cases treated until the erythrocyte microsedimentation rate had returned to normal, we find a decreasing tendency in the recurrence rate, a feature which is especially noticeable when we study the conditions after the passage of 7 years from the end of the initial attack. This drop in the number of cases with a first recurrence is, however, not so large as to border on statistical probability (the difference between the year groups 1922—1931 and 1932—1937, for the recurrence rate within 7 years, is 6.9 % $\pm$ 5.3).

Thus, although a certain reduction in the rate for the first recurrence is noticeable in the material, yet this drop is comparatively

Table 15.

Recurrence rate (with respect to first recurrence after initial attack) within 4 and 7 years in different year-groups, calculated on the basis of the total number of cases, not including cases with chorea as the initial manifestation of rheumatic fever.

The figures within brackets include the 4 cases admitted to the hospital in 1941 or 1942 but treated at home for their initial attack during the years 1938—1940 (see comment in table 16).

Initial attack in year-group:	Total cases surviving initial attack	No. of cases with a first recurrence within 4 years	Percentage of total cases	No. of cases with a first recurrence within 7 years	Percentage of total cases
1922—25 . .	56	25	44.6 ± 6.6	33	58.9 ± 6.6
1926—29 . .	124	34	27.4 ± 4.0	44	35.5 ± 4.3
1930—33 . .	92	23	25.0 ± 4.5	36	39.1 ± 5.1 [1]
1934—37 . .	88	32	36.4 ± 5.1	34	38.7 ± 6.6
1938—40 . .	119 (123)	29 (33)	24.4 ± 3.9 (26.8 ± 4.0)	—	—
1922—31 . .	231	73	31.6 ± 3.4	100	43.3 ± 3.2
1932—40 . .	248 (252)	70 (74)	28.2 ± 2.9 (29.4 ± 2.9)		
1932—37 . .	129			47	36.4 ± 4.2

small, and here also, therefore, statistical significance or probability cannot be established. Considered together with the gradual change in the character of the disease which has already been demonstrated, it may perhaps be presumed that this change also is not wholly a matter of chance. (Cf. also chap. 5, on the recurrence rate.)

Turning now to possible reasons for this change in the nature of the disease which has thus been demonstrated, I refer again to the previously mentioned enlightenment that has been gaining

[1] The rate of recurrence within 7 years for the year-group 1930—1931 amounts to 45.1 % (23 first recurrences among 51 cases) and for year-group 1932—1933 to 31.7 % (13 first recurrences among 41 cases).

ground, especially during the last quarter of a century, regarding the importance of early and consistent treatment of children suffering from rheumatic diseases. The noticeable change that my results would seem to demonstrate, with reference to the more recent year-groups as compared with the 1922—1925 period, may partly be explained by the altered attitude to the necessity for hospital attention, resulting from the increased realization of the gravity of the disease. However, this can hardly constitute the whole explanation.

The previously suggested tendency to a reduction in the recurrence rate, and the strikingly reduced total mortality, might, of course, be explained by such factors as longer and more consistent care in the individual cases, and in particular a longer period in bed and a closer observation of the subsequent course in the form of more frequent control examinations, factors that have become more and more common in the present material. But evidence against this being the only and the true cause is the fact that the primary mortality dropped in the same way as the total mortality. Other factors also must obviously play a contributory rôle, and one of the first to come into our minds is a possible change in treatment. The only change, as regards the treatment of the actually active disease, that has been made, is an extension of the treatment time which, especially with respect to the second half of the material, has as a rule been extended to include bed rest until the microsedimentation rate drops to normal values and becomes stabilized at that level. The primary mortality cannot, of course, have been influenced by this extension in the length of treatment, however.

The only factor likely to have had an effect on the primary mortality also, as well as on other similar primary factors (e.g. the risk for pericarditis at the initial attack) is that, because of the increasing knowledge regarding the disease, medical advice might have been sought and the patients remitted to the hospital at an earlier stage in recent years than was the case before. The well-known difficulty of effectively keeping children in bed at home may have contributed towards causing patients who had been treated at home for a fairly long period before their admission

to the hospital to be at a disadvantage as compared with patients who were kept consistently in bed from the very first stages of the disease, a measure which in general is equivalent to hospital care.

In order to ascertain whether any change had occurred as regards the duration of the illness before the admission of the children to the hospital (i.e. regarding the number of days from onset to admission) I endeavoured on the basis of the hospital records to determine with respect to every case (exclusive of the chorea cases), the number of days each patient was treated at home before coming to the hospital, irrespective of whether they had stayed in bed for this period or had been up and about. The result is shown in table 16. (The number of treatment days at home was originally divided up into much smaller units of 3, 4, or 5 days, in addition to which other combinations than those shown in the table were examined, but no different, or better, information was obtained.) Those patients who had been treated at home during the whole of the initial attack, irrespective of whether they had been under medical observation or not, have been grouped together with those who had been treated at home for their rheumatic fever for more than three weeks before being admitted to the hospital — the treatment time at home among the latter was, in fact, extremely varying, and only in a few cases was it around 3—4 weeks.

As may be seen from this table, the number of patients admitted during the first week of their illness amounts to nearly 50 per cent of the total number of rheumatic fever patients, and this proportion is the same in all year-groups. On the other hand, the percentage incidence of cases first admitted to the hospital in the second to third week after the onset of the infection shows a rising tendency in the later part of the period under investigation, while the opposite tendency is observed with regard to the patients treated at home for more than 3 weeks or during the whole of their initial attack (see fig. 10). This tendency is statistically probable, as will be seen from the results of the χ^2-test. (The same tendency is apparent from an analysis of the cases treated at home during the whole of the initial attack

Table 16.

Showing the total number of cases (not including cases with chorea as the initial manifestation) classified according to the number of days of illness at home before admission to hospital.

Relation between number of cases admitted to hospital on 8th to 21st day and total number of rheumatic fever cases in the different year-groups tested with the χ^2-method for heterogeneity. Relation between total cases and cases treated at home for more than 21 days, including those treated during the whole initial attack, tested in the same way.

Calendar year at onset	Total cases	Number of cases treated at home for the following lengths of time before admission to hospital					
		0—7 days		8—21 days		> 21 days[3]	
		No. of cases	%	No. of cases	%	No. of cases	%
1922	19	9		2		8	
1923	20	11		2		7	
1924	14	5	35 50.0	4	9 12.9	5	26 37.1
1925	17	10		1		6	
1926	37	22		9		6	
1927	25[1]	12		2		10	
1928	37	18	72 52.1	8	27 19.6	11	38 27.5
1929	39	20		8		11	
1930	38[1]	17		10		10	
1931	20[1]	5		8		6	
1932	21[1]	9	47 44.8	6	29 27.6	5	26 24.8
1933	26	16		5		5	
1934	35[1]	16		8		10	
1935	17	18		5		4	
1936	18	7	43 46.4	6	21 22.6	5	28 30.1
1937	23	12		2		9	
1938	39+1	24		12		3+1	
1939	59+1	27	66 51.5	19	38 29.7	13+1	24[2] 18.8
1940	26+2	15		7		4+2	
				$\chi^2 = 9.4$, P approx. = = 0.05, d. f. 4		$\chi^2 = 11.9$[4], P < 0.05, d. f. 4	

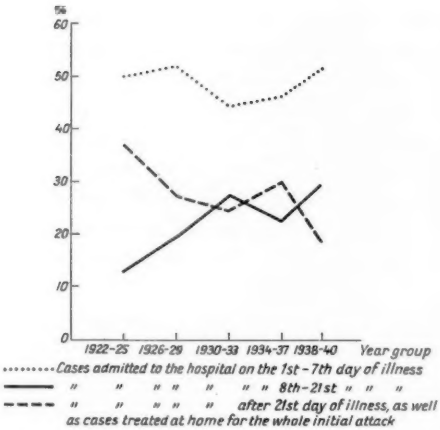


Fig. 10. Showing percentage of patients in different years grouped according to the number of days of illness before admission to hospital.

and of those treated at home for the initial attack for more than 21 days, but because of the smallness of the series in each of these groups, no statistically probable difference can be obtained.)

There is obviously a definite tendency, in more recent years, not to let the children lie too long at home with this disease. There would seem to be little doubt that this circumstance must have contributed in some measure towards producing the more favourable prognosis observed in more recent years, but the extent to which it has contributed is probably impossible to establish.

---

[1] In one case it was impossible to determine length of time patient had been treated at home before admission to hospital.

[2] 4 cases (indicated in the table by the sign +) admitted to the hospital in 1941 and 1942 for a recurrent attack and treated at home for the initial attack are included in this figure.

[3] In this column are included all the cases treated at home during the entire initial attack.

[4] After adding the 4 cases mentioned in table 4 (indicated in the above table by the sign +), which had been treated in 1941 and 1942, we get a χ^2 value = 8.9 and $0.05 < P < 0.10$, d. f. 4.

Other factors likely to have played a part in improving the prognosis are, perhaps, an improved standard of living, improved hygiene, food, and living conditions, more sensible clothing, a more open-air life, and so on, all of which must have helped to increase the powers of resistance of the population, not least those of the children. But quite apart from the possibility of a change in the above-mentioned conditions, the well-known alteration in the *genus epidemicus*, observed in connection with a number of other diseases, may, of course, also have played a rôle here, and may, perhaps, be the true explanation of the gradually improving prognosis of rheumatic fever. All these suggestions, however, can at present be nothing more than speculations.

Because of these differences between earlier and more recent cases, the material will from now on be divided into two halves of approximately the same size, the one group comprising patients contracting rheumatic fever during the years 1922—1931, and the other comprising patients from the years 1932—1940. In addition, corresponding figures for the whole material as one unit will also as a rule be given. When this system of classification yields figures too small to be of value for illustrating certain conditions it will be disregarded without any special mention being made of the fact.

CHAPTER 4.

Mortality.

Causes of death.

Of the total 689 rheumatic fever patients treated during the years 1922—1940, 127 (18.4 per cent) had died before the middle of 1944.[1] In 111 cases (16.1 per cent) death was caused by the rheumatic cardiac affection or its aftermaths, while the remaining 16 patients died from other causes. These other causes are given in the following list:

Disease	No. of cases
Tuberculosis	5
Pneumonia	2
Accidents	2
Carbon monoxide poisoning	1
Multiple sclerosis	1
Meningitis	1
Chronic nephritis	1
Endarteritis obliterans + thrombosis and pulmonary embolism	1
Diphtheria	1
Scarlatina	1

The patient who died of scarlatina (Record no. 549/1938) had been taken ill 14 months before death with a severe, initial attack of rheumatic fever associated with pericarditis, for which condition he was treated for 135 days in hospital. In all probability, the cardiac damage arising in connection with this disease was a contributory cause of death, although it is impossible to decide how large a part it played in the outcome. Consequently, the case has not been included among those where death was considered to be due to the rheumatic infection.

[1] It is assumed here that the 6 patients with whom no actual contact could be made are still alive.

Table 17.

Number of years from onset to death (for cases where death was due to some other disease).

No. of years	No. of cases	No. of years	No. of cases
$<\frac{1}{2}$	1	9—10	1
1—2	3	12—13	3
3—4	1	13—14	1
6—7	1	15—16	1
7—8	1	17—18	1
8—9	1	19—20	1

On the other hand, the patient who died of diphtheria (Record no. 1100/1930) had had a mild attack of rheumatic fever a little more than 1 $^1/_2$ years previously, with moderately severe arthritic symptoms and signs of mild carditis in the form of myocarditis with a prolonged P—Q interval (P—Q: 0.20 sec.; this value dropped within one week to 0.16 sec.), but no definite signs of valvulitis. When the patient was admitted to the hospital for epidemic diseases the physical examination yielded no abnormal findings from the heart.

Among the 16 cases where death was due to some other disease or to accidents, information as to the condition of the heart in connection with or immediately prior to the last illness was obtained in 4 cases (in 3 the heart was normal and in the fourth there was probable vitium cordis). In 5 other cases, an autopsy yielded the following results, viz. heart normal in two cases, myocardial disorder in two cases (with hypertrophy of the cardiac muscle in one of them) and mitral deformity in one case.

The number of years these 16 patients lived after the onset of rheumatic fever will be seen in table 17.

Table 18 shows the 111 patients who died of rheumatic fever or its aftermaths. In 3 (2.7 per cent), possibly 4, cases blood culture yielded *Streptococcus viridans* bacilli on one or more occasions. It is possible, though far from certain, that this figure would have been higher if more frequent blood cultures had

Table 18.

Cause of death and classification according to the nature of the initial attack in the 111 cases dying more or less as a direct consequence of rheumatic fever.

Cause of death	Nature of initial attack			Total
	Chorea	Arthritic symptoms	Carditis alone	
Active carditis . . .	7 [2]	71 [4]	18 [6]	96
Cardiac disease [1] . .	4 [3]	6	1	11
Endocarditis lenta .	—	3 [5]	1(?) [7]	3(+1?)
			Total	111 cases

been carried out in each case. In many patients, blood cultures were not made at all; this was especially the case among patients who had been treated under the care of a physician in their own homes for later attacks, and had died there.

In one case gliosis of the brain was stated to be a contributory cause of death. The child in question, a girl (Record no. 944/1929), was taken

[1] Diagnosis according to death certificate: Vitium cordis or chronic myocarditis. Not known whether active carditis was conjointly present.

[2] Pulmonary embolism or pulmonary infarction was stated to be a contributory cause of death in two cases. One case had gliosis of the brain also.

[3] Pulmonary infarction a contributory cause of death in one case.

[4] In 7 cases bronchopneumonia was a contributory cause of death; in 4 cases pneumonia; in 2 cases pulmonary tuberculosis; in 4 cases pulmonary infarction (followed by pneumonia in one case); in 1 case pulmonary abscess and empyema; in 1 case biliary cirrhosis; in 1 case mongolism.

[5] (Record nos. 603/1928, 22/1933, and 1136/1936.) Initial attack in all cases 15, 8, and 8 years respectively before the recurrent attack (the only one), which was characterized by the presence of *Streptococcus viridans* in the blood and caused the death of the patients.

[6] In 4 cases bronchopneumonia was also given as a contributory cause of death; in 2 cases pneumonia.

[7] A blood culture revealed the presence of streptococci (type not stated). The patient (Record no. 318/1929) fell ill 17 days before admission to hospital with an initial attack of carditis. She was treated for 4 days at the hospital and then died.

ill at 4 years of age with chorea followed by arthritic symptoms in the same attack, and was treated at home for 6 weeks. Two years later she had a recurrence, with arthritic symptoms, choreiform movements appearing again after some time. At the end of two months' illness, one month of which was spent at home, the choreic restlessness increased in violence and shortly before death passed over into general apathy. Postmortem examination revealed, in addition to pancarditis, cerebral gliosis. ("Dura somewhat thickened, membranes soft, in places showing signs of milkiness. Brain pale. Lateral ventricles rigid, with a thickened ependyma; in a few areas, small stringy adhesions between opposing surfaces. Brain stem shows increasing consistency in a downward direction, most pronounced in the oblongata, which is almost leathery. The cut surface pale.")

Age at onset of infection and at death.

The age at onset in children who died of their rheumatic infection or from its aftermaths is shown in table 19. The comparatively small number of cases in each age-group allows of no significant conclusions being drawn from this table. There is no striking difference between the sexes as regards the number of deaths, but a lag is to be observed, as before, in the girls' curve as compared with that of the boys.

If, instead, we distribute the deaths on age-groups of 3 years and then calculate the percentage incidence of the mortality (table 21), we find that as the age at onset increases there is a distinct drop in the mortality in relation to the incidence of rheumatic fever (see fig. 11), a decrease which, from the statistical standpoint, can be said to be probable with respect to the primary mortality and bordering on very probable with respect to the total mortality. A comparison of the 1—3 year and 13—15 year age-groups shows a difference which is statistically very probable.

A study of table 21 reveals that, in this material, 33 per cent of the patients died, if the initial attack occurred between the ages of 1 and 3 years, 20 per cent died if it occurred between 4 and 6 years, 15 per cent if it occurred between 10 and 12 years, and 8 per cent if it occurred between 12 and 15 years. If the limit for the age of childhood be set at 16 years, the mortality from

Table 19.

Age at onset.

Age in years	Males	Females	Both sexes
2— 3 .	1	1	2
3— 4 .	5	2	7
4— 5 .	2	2	4
5— 6 .	5	3	8
6— 7 .	7	3	10
7— 8 .	8	10	18
8— 9 .	5	6	11
9—10 .	4	7	11
10—11 .	3	4	7
11—12 .	8	8	16
12—13 .	5	3	8
13—14 .	2	3	5
14—15 .	2	2	4
Total	57	54	111

Table 20.

Age at time of death.

Age in years	Number of cases Both sexes	
2— 3 .	—	6
3— 4 .	6	
4— 5 .	4	
5— 6 .	3	13
6— 7 .	6	
7— 8 .	4	
8— 9 .	12	24
9—10 .	8	
10—11 .	5	
11—12 .	17	32
12—13 .	10	
13—14 .	7	
14—15 .	9	22
15—16 .	6	
16—17 .	5	
17—18 .	2	9
18—19 .	2	
19—20 .	2	
20—21 .	1	
23—24 .	1	
26—27 .	1	
Total	111	

rheumatic fever during the first 5 years of life (27.6 per cent) is approximately twice as high as it is during the last 5 years (14.2 per cent).

If the cases with chorea as the initial manifestation are left out of account (because of the low death rate among these cases), the above-mentioned figures, as well as all the others in table 21, will be altered only very inconsiderably.

Table 21.

Total cases where death was due to rheumatic fever, divided into age-groups of 3 years according to the age at onset.

Relation between number of deaths and cases with rheumatic fever in the different age-groups tested with the χ^2-method for heterogeneity.

Age-group in years	Total rheumatic fever cases in each group	No. of deaths at initial attack	Percentage of total	Total deaths	Percentage of total
A. 1— 3 . . .	27	6	22.2	9	33.3
B. 4— 6 . . .	111	11	9.9	22	19.8
C. 7— 9 . . .	235	16	6.8	40	17.0
D. 10—12 . . .	202	15	7.4	31	15.3
E. 13—15 . . .	114	5	4.4	9	7.9
Total	689	53		111	
		$\chi^2 = 10.8$, $P < 0.05$, d. f. 4		$\chi^2 = 13.0$, $0.01 < P < 0.02$, d. f. 4	
				Diff._{A-E}: 25.4 ± 9.4	

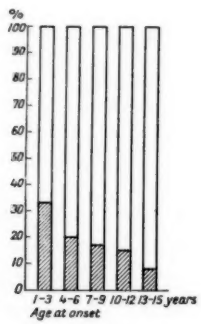


Fig. 11. Percentage of deaths among total number of rheumatic fever cases, grouped according to age at onset.

The age at death will be seen in table 20. If we combine these cases into age-groups of 3 years, as before, the number of deaths is seen to increase gradually up to a peak at 10—12 years of age, after which it decreases again with approximately the same incline. Here also, however, the number of cases in each age-group is so small that no significant inferences can be made from the figures obtained.

Duration of disease in fatal cases.

As already mentioned, the mortality from rheumatic fever or its aftermaths shows a strong, gradual, statistically significant decline during the years 1922—1940 (see fig. 9 p. 88). If, therefore, we divide up the material in the way previously indicated, into the year-groups 1922—1931 and 1932—1940, and investigate, both in each of these two groups and in the whole material, how long a time had elapsed between the onset of rheumatic fever and death, we get the results shown in table 22 and the histograms in figure 12. From these histograms we obtain a strikingly clear picture of what a strong reduction has taken place in the mortality in recent years and also of how this decrease is fairly evenly distributed in relation to the number of years that have elapsed since the initial attack. (N. B. The first two co-

Table 22.

Number of years from onset to death.

The table shows the number of cases in the respective year-groups.

	Year from time of initial attack																		Total deaths
	0—½ V	½—1	1—2	2—3	3—4	4—5	5—6	6—7	7—8	8—9	9—10	10—11	11—12	12—13	13—14	14—15	15—16	16—17	17—18	
1922—31 . . .	31	8	6	4	8	3	3	2	3	2	3	1	—	—	1	—	1	—	1	77
1932—40 . . .	15	2	1	2	2	1	—	—	1	1	—	—	—	—	—	—	—	—	—	25
Whole material[1]	46	12	9	8	10	4	4	2	5	3	3	1	—	—	1	—	2	—	1	111

[1] Incl. cases with initial attack before 1922.

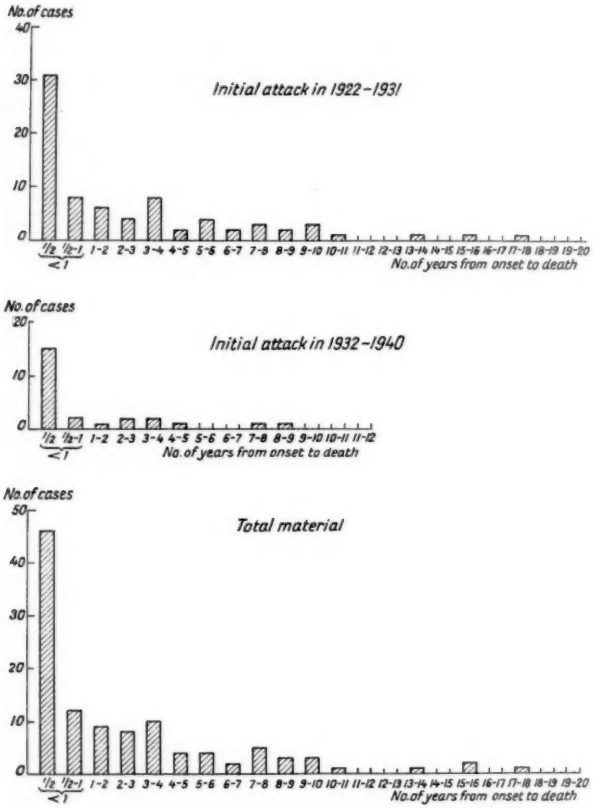

No. of cases
Initial attack in 1922–1931
No. of years from onset to death
No. of cases
Initial attack in 1932–1940
No. of years from onset to death
No. of cases
Total material
No. of years from onset to death

Fig. 12. Number of years between onset and death.

lumns in the histograms embrace the first year after the initial attack.)

It should be pointed out here, that for the first four years the year-groups 1922—1931 and 1932—1940 are fully comparable. As regards the 1932—1940 year-group, it is, of course, quite conceivable that a few more deaths would have occurred if all cases had been observed for as long a time as the shortest obser-

vation time for the 1922—1931 year-group (i.e. 13 years). If it be assumed that proportionately as many deaths will occur in the 1932—1940 year-group, taken in relation to the first 4 years, as in the 1922—1931 year-group, with an average observation time of 13 years (= the shortest observation time for the 1922—1931 year-group) in each group, a simple calculation will reveal that with an observation time of at least 13 years in the 1932—1940 year-group the number of deaths will increase by only two cases (= 2.09 cases). It can therefore be said that the two histograms are fully comparable.

Further examination of table 22 and the histograms in figure 12 reveals that the mortality has dropped to half, as far as the first half-year of the two groups is concerned, but that the remaining mortality for the first 4 years has decreased to an even greater degree, from 26 cases to 7; in other words, it has dropped to nearly one-fourth, and this seems also to apply to the 13 years following the initial attack.

As may be seen from the table and the histogram covering the total number of deaths, the mortality is highest in the first year after onset and in this year it is highest in the first half. During the second half of the first year and the following three years it remains more or less at the same level, and in the fourth year it drops to a minimum.

As has been mentioned in the survey of earlier publications, the mortality seems to be highest during the years immediately subsequent to the initial attack, according to some authors, while others, especially the American investigators, have found that the death rate does not reach its maximum until after the passage of some years from the onset of the infection. In my material, there is no doubt whatsoever that the highest mortality is directly connected with the initial episode, and that it subsequently decreases to a noticeable extent. In all probability, the mortality will rise again later, but not until the heart lesions produced by the disease begin to manifest themselves in the form of increasing congestive heart failure as the patients grow older. And this will undoubtedly not occur until

after many more years have elapsed from the initial attack than the number of years embraced by this investigation. The observation time, dating from the initial attack, in those publications in which the mortality curve was found to rise in the direction of the later years, is, however, no longer than in my material, as a rule it is shorter, and in this respect therefore, the different materials ought to be comparable. It is obvious that we are faced here with a difference in the essential nature of the disease which in all probability is referable chiefly to the different geographical locations of the materials or some other similar factor. The correct explanation of the difference is, however, impossible to supply here, and very probably the only way to solve the problem would be to make investigations on the spot.

Autopsy findings.

In order to be able to use postmortem material for making inferences of general applicability regarding, for instance, the degree of the cardiac affection resulting from rheumatic fever, it is essential that the material should be really representative of all the cases that have had this disease. In other words, one must study the autopsy findings not only in the severe cases with a fatal outcome at an early stage, but also in those surviving both one and several attacks. This means that one either has to wait till the person has grown old and died, or else that autopsy material must be obtained from persons dying as the result of accidents. But to wait till the person dies of old age means that so many other factors may affect the condition of the heart that an autopsy will yield a result difficult to evaluate. And on the other hand, the chances of obtaining a sufficiently large material from persons who have died as the result of an accident, are fairly small.

Material filling these requirements was not at my disposal in connection with this investigation. Nor does the postmortem series mentioned in this section include all the patients from the whole material who died. Among the 127 patients who died, 75 were submitted to postmortem examination. Histologic exam-

Table 23.

Different types of cardiac affection found at autopsy in 75 patients.

	No. of cases not examined post mortem	No. of autopsies	No. of cases with following valves affected					Affection of myocardium only	No cardiac affection	No. of autopsied cases with pericarditis
			Mitral valve	Aortic valve	Mitral and aortic valves	Mitral, aortic, and tricuspid valves	All valves			
Males .	32	36	8	1	17	5	3	2	—	22
Females	20	39	13	2	16	3[1]	3	—	2	30
Total	52	75	21	3	33	8[1]	6	2	2	52
			(28.0%)	(4.0%)	(44.0%)	(10.7%)	(8.0%)	(2.7%)	(2.7%)	(69.3%)

ination of, for instance, the heart, was not as a rule carried out; usually, only the gross appearances were noted at the postmortem examination. Only 5 patients who died of other diseases are included in this autopsy material. For the rest, it is composed of patients who died more or less as an immediate consequence of their rheumatic infection.

In table 23 there are assembled the findings from the heart that were made in connection with these 75 autopsies. As may be seen, only 2 patients had no cardiac changes at all. Lesions in the pericardium indicative of pericarditis that was either active at the time of death or was suffered at an earlier period were present in 52 cases (69.3 per cent). A grossly visible nidus suggestive of myocarditis was observed in most of the cases. In 2 cases the only lesion was myocardial damage.

One of the two last-named patients died of endoarteritis obliterans in association with thrombosis and pulmonary embolism, and displayed at autopsy a pea-sized induration in the myocardium; the other died of pulmonary tuberculosis, and postmortem examination revealed a "moderately enlarged heart with slight bilateral hypertrophy, smooth valves, and abnormally hard, slightly streaky, muscle".

[1] In 1 case the tricuspid and mitral valves only were affected.

As regards the valvular affection, this is illustrated best by a reference to the table. It is worthy of note that in not a single one of the cases were the tricuspid and pulmonary valves found to be the only seat of change, and in only 3 instances was this the case with the aorta, while the mitral valve was the only seat of damage in a little over one-fourth of the cases.

As will be mentioned further on (p. 162), the postmortem examination revealed the presence of pericarditis in 17 cases where, to judge from the hospital records, no such signs had been observed at the clinical examination. It is, however, possible that pericarditis may have been suspected in some cases although it was not mentioned in the records.

Mortality rate in relation to type of initial manifestation and to number of attacks passed through.

What rôle is played by the type of the initial manifestation of rheumatic fever in the mortality rate? In order to elucidate this question I constructed table 24, in which the material has been divided up according to the nature of the initial manifestation. It will be observed that when the initial attack took the form of chorea, only one single death occurred in connection with the initial attack. On the other hand, the mortality from recurrent attacks, in this chorea group, does not diverge to any noticeable extent from the average mortality rate for all cases.

The child that died in connection with the initial attack of chorea (Record no. 1105/1934), a 5 year old girl, had had mild symptoms of choreic type for 2 $^1/_2$ months without medical advice being sought. Three weeks before her admission to the hospital she had had fever for 4 days, and according to her parents she had also had a rash resembling a measle rash all over her body, but particularly noticeable on her buttocks and face. About a week later her temperature rose again. During the whole of this time she suffered from choreiform grimacing. On admission she was found to have severe chorea possibly in association with pericarditis. Death occurred one month later, due to carditis.

A remarkable feature is the strikingly high mortality, as compared to the average mortality, in the group of cases with

Table 24.

Deaths grouped according to the nature of the initial attack and the number of deaths occurring at the first or a subsequent attack.

(Only patients who died of rheumatic fever or its aftermaths are included.) The table shows the number of cases. The percentage figures in relation to the total number of cases in each group are shown within brackets.

Nature of initial attack	Total cases	Deaths at initial attack	Deaths at a later stage	Percentage of deaths among those surviving the initial attack
Chorea	127	1 (0.79 %)	10 (7.9 %)	7.9 %
Arthritic symptoms	521	39 (7.5 %)	41 (7.9 %)	8.5 %
Carditis	41	13 (31.7 %)	7 (17.1 %)	25.0 %
Total	689	53 (7.7 %)	58 (8.4 %)	10.2 %

carditis alone at the initial attack; this observation applies both to the mortality at the initial attack and the mortality at a later stage. This high death rate can hardly be satisfactorily explained by assuming that the milder cases with carditis alone were never brought to the hospital for medical attention, although it seems not unlikely that this possibility may have played some part in producing the conditions observed.[1] The slightly lower mortal-

[1] It is, of course, conceivable that in some of the patients who had carditis alone at the initial attack there were arthritic symptoms also, but of such a mild nature that they were soon forgotten and thus not mentioned when the case record was being written up. If now, the absence of all arthritic symptoms were to mean a more pronounced affinity of the rheumatic disease to the heart, then the patients belonging to the transition group (between the group with arthritic symptoms at onset and that with carditis alone) and remembering these arthritic symptoms at the time the personal history was recorded, ought to show an increased mortality as compared with the remaining cases with arthritic symptoms. I therefore went through all the case records containing mention of arthritic symptoms at the initial attack, and picked out from among these the cases that had very mild or barely distinguishable manifestations from the joints (i. e. cases with subjective symptoms only — never objectively observable signs — when these had lasted for 4—5 days at the most, were characterized by transiency, and had caused no restriction of movement, but possibly pain on extreme movement). By this method, 58 cases (one of which

ity rate apparent in the table in respect of later attacks (as compared with the mortality at the initial attack), in cases with carditis alone at the initial attack, is probably due to chance, seeing that the number of cases is so small.

Table 25 shows the mortality in relation to the number of attacks of rheumatic fever the patients have passed through. As may be seen, there is a progressive rise in the mortality with the appearance of fresh attacks, this rise being statistically significant ($\chi^2 = 14.1$, $P < 0.001$, d. f. 2) for all cases as regards the first three attacks; on the other hand, a comparison between the first and second attack only gives a statistically probable difference, while between the second and third attack the difference does not reach statistical probability. (The small number of cases with more than three attacks makes it inadvisable to draw any definite conclusions from this group.)

It would seem here to possess a certain interest to examine the mortality in relation to the number of attacks the patients have passed through as evidenced in the two halves of the material (see table 26). It will be seen that in the 1922—1931 year-group there was a gradual rise in the mortality with each new attack passed through, a rise that seems more noticeable than the corresponding rise for the whole material. (A χ^2-test with respect to the first three attacks also yields the result $\chi^2 = 9.77$ and $P < 0.01$, d. f. 2.) Considering, on the other hand, the group of patients who had their initial attack in 1932—1940, the mortality seems to be fairly uniform with respect to the first and second attack while with the appearance of the third attack it seems to

---

was not submitted to the follow-up examination) were obtained. The total number of deaths in this group was found to be 10 cases (17.2 per cent) as compared with 70 cases (15.1 per cent) among the remaining 463 cases with arthritic symptoms. The primary mortality on the other hand, amounted to 15.5 per cent (9 cases) in this group with very mild joint manifestations, while the corresponding figure for the remaining cases with these arthritic symptoms was 6.7 per cent (31 cases). The difference as regards primary mortality between these two groups with joint manifestations is not statistically probable, however, and no inferences can therefore be made from these figures, although the tendency seems to point in the direction indicated at the beginning of this footnote.

Table 25.

Death rate with respect to different attacks.

(Only patients who died of rheumatic fever or its aftermaths are included.) Eighteen cases which were not re-examined and where it was impossible to obtain reliable information as to whether there had been later attacks or not, are missing from the total number of cases. As regards the figures in brackets, the numerator indicates the number of deaths and the denominator the number of cases with the attack shown in the column heading.

Primary manifestation	Initial attack	Second attack	Third attack	Fourth or fifth attack
Cases with arthritic symptoms and cases with carditis alone	9.5 % ± 1.3 (52/549)	13.9 % ± 2.3 (32/230)	18.9 % ± 4.6 (14/74)	7.1 % ± 4.9 (2/28)
Chorea	0.8 % ± 0.8 (1/122)	6.5 % ± 3.1 (4/62)	20.8 % ± 8.3 (5/24)	14.3 % ± 13.2 (1/7)
All cases	7.9 % ± 1.0 (53/671)	12.0 % ± 1.9 (35/292)	19.4 % ± 4.0 (19/98)	10.8 % ± 5.1 (4/37)

increase. (Because of the small number of cases that had a third attack no final conclusions can be drawn from the lastmentioned circumstance, however.)

Taking each attack separately (table 26), it is found that the difference between the two year-groups in respect of the death rate for the first attack is statistically probable, and for the second attack statistically significant, while for the third attack the small series of cases does not allow of a statistically probable difference being established, although here also there is a fairly marked difference. In any case, it seems fairly evident that there is a considerable reduction in the mortality rate for the 1932—1940 year-group as compared with the 1922—1931 year-group, even when each attack is considered separately. (Cf. chap. 3.)

As regards the reason for this improvement in the prognosis in the 1932—1940 year-group possible explanations have already

Table 26.

Death rates with respect to different attacks, divided into year-groups according to the calendar year at onset.

(Only patients who died of rheumatic fever or its aftermaths are included. The table does not include the 18 cases which were not re-examined and where no reliable information as to possible later attacks could be obtained, nor the 30 cases where the initial attack had occurred before the year 1922. As regards the figures in brackets, the numerator indicates the number of deaths and the denominator the number of cases with the attack shown in the column heading.

Year-group	Initial attack	Second attack	Third attack	Fourth or fifth attack
1922—1931	10.5 % ± 1.7 (35/334)	16.0 % ± 2.9 (25/156)	25.0 % ± 5.8 (14/56)	15.0 % ± 8.0 (3/20)
1932—1940	5.5 % ± 1.8 (17/307)	3.7 % ± 1.8 (4/108)	14.3 % ± 6.6 (4/28)	— (0/7)
Difference between year-groups	4.9 % ± 2.1	12.3 % ± 3.5	10.7 % ± 8.9	—

been advanced. It may perhaps be stated here, however, that the fact that the mortality is approximately the same at the first and second attacks, in the 1932—1940 period, as compared with a higher mortality for the second attack in the 1922—1931 period seems to me to indicate that the longer period in bed in connection with the initial attack, and the prolonged and more intensive after-control of cases having the initial attack in the years 1932—1940, have helped to place these patients in a more advantageous position on the appearance of fresh attacks, the risk of death at later attacks being thereby reduced.

CHAPTER 5.

Recurrence.

Definition.

By recurrence is meant in the present work a return of the disease after the disappearance of all the clinical signs and symptoms (including the increase in the microsedimentation rate of the red cells, and as regards chorea, all choreiform movements even in connection with excitement). Thus an exacerbation of the disease in a patient who may, certainly, have been in a less active stage but in whom the microsedimentation reaction has not yet gone down to normal values, is not here counted as a recurrence. Nor have I counted as a recurrence an exacerbation of the symptoms in a patient in whom all the subjective symptoms and objective signs have disappeared with the exception of an abnormally high microsedimentation rate, indicating that the disease has not yet definitely been overcome. Thus if towards the end of his illness such an individual has contracted pharyngitis, and in connection with this, signs of reactivation of the rheumatic infection have been observed, this has not been described as a recurrence. On the other hand, a relapse after a symptom-free interval, but with a different manifestation than the first time, e. g. an initial attack of chorea[1] with a relapse in the form of arthritic symptoms[1] or signs of carditis alone, and *vice versa*, has also been counted as a recurrence. If, however, in those cases with arthritic symptoms as the initial manifestation choreiform movements appear in the course of this attack, or *vice versa*, this has not been counted as a recurrence.

[1] With or without carditis (as previously mentioned — see footnote on p. 59).

Table 27.

Recurrence rate in relation to the nature of the initial attack.

The percentage incidence has been calculated in relation to the total number of survivors.

Nature of initial attack	Total cases	No. of cases surviving the initial attack	Patients surviving initial attack	
			No recurrences	One or more recurrences
Chorea	122	121	59 (48.8 % ± 4.5)	62 (51.2 % ± 4.5
Arthritic symptoms	510	471	256 (54.4 % ± 2.3)	215 (45.6 % ± 2.3
Carditis alone . .	39	26	11 (42.3 % ± 9.7)	15 (57.7 % ± 9.7
All cases	671	618	326 (52.8 % ± 2.0)	292 (47.2 % ± 2.0

Recurrence in relation to various initial manifestations.

A study of table 27 shows that in this material (671 cases) an average of 47.2 % ± 2.0 of patients surviving the initial attack subsequently had one or several recurrences of some form of rheumatic fever. It also seems as if the number of cases that had any recurrence at all is somewhat greater among those cases that had chorea at the initial attack than among those having arthritic symptoms; but the difference is not statistically probable. In those cases where the initial attack took the form of carditis alone, this observation applies in a still higher degree.

From the purely clinical point of view it would definitely seem as if, in patients who have chorea as the first evidence of the disease, subsequent recurrences also usually take the form of chorea, although not infrequently, also, patients may have a recurrence with some other manifestation of rheumatic fever. This is also found to be the case in my material (see table 28). Among the patients with chorea as the initial manifestation who afterwards had a recurrence, this recurrent attack took the form

Table 28.

Type of recurrent attacks in relation to the initial manifestation.

Nature of initial attack	No. of cases with one or more recurrences	Number of cases with the following types of recurrent attack. (Where several types are mentioned different attacks are referred to in most instances)						
		Chorea	Arthritic symptoms	Carditis alone	Chorea. Arthritic symptoms	Chorea. Carditis alone	Chorea. Arthritic symptoms. Carditis alone	Arthritic symptoms. Carditis alone
Chorea[1] . .	62	35 (56.5 %)	8 (12.9 %)	5 (8.1 %)	9 (14.5 %)	4 (6.5 %)	—	1 (1.6 %)
Arthritic symptoms[2]	215	21 (9.8 %)	147 (68.4 %)	22 (10.2 %)	13 (6.1 %)	2 (0.9 %)	—	10 (4.7 %)
Carditis[3] .	15	2 (13.3 %)	7 (46.6 %)	4 (26.7 %)	1 (6.7 %)	1 (6.7 %)	—	—
Total cases	292	58 (19.9 %)	162 (55.5 %)	31 (10.6 %)	23 (7.9 %)	7 (2.4 %)	—	11 (3.8 %)

of chorea in somewhat over half the cases (56.5 per cent), while in 21.0 per cent, besides an attack of chorea, there was also occasionally an attack of arthritic symptoms or of carditis alone. Among the patients with recurrences, in whom chorea was the initial manifestation, chorea thus appeared as a recurrence in some attack in 77 per cent. In no fewer than 43.5 per cent of the patients with chorea at the initial attack who had a recurrence, arthritic symptoms or carditis alone were present at least in one attack.

[1] In this group there were 3 patients (2 without recurrence, 1 with recurrence) with chorea as the primary manifestation, followed by arthritic symptoms during the initial attack.

[2] In this group there were 22 patients (13 without recurrence, 9 with recurrence) with arthritic symptoms as the primary manifestation, followed by chorea during the initial attack.

[3] In this group there were 4 patients (2 without recurrence, 2 with recurrence) with carditis as the only primary manifestation, followed by chorea during the initial attack.

Similarly, it would seem that the patients with arthritic symptoms at the initial attack as a rule have arthritic symptoms in their recurrent attacks; but in a few instances, of course, other forms of rheumatic fever also appear. In this group two-thirds (68.4 per cent) of the patients who had recurrent attacks had only arthritic symptoms in subsequent attacks, and no fewer than 89 per cent of the recurring cases had arthritis in some attack. Chorea in a recurrent attack following an initial attack with arthritic symptoms occurred, on the other hand, in only 16.8 per cent (= 36 cases) or one-sixth of the cases.

Roughly the same percentages apply to those patients who, during the initial attack of the disease, did not manifest any signs of arthritis or chorea at all, though the small number of cases with carditis alone that later had a recurrence (15 cases) does not permit of any detailed analysis or comparison.

Considering the series as a whole, it will be found that of the cases experiencing recurrences one-third, or 30.2 per cent, have chorea at some time, two-thirds, or 67.2 per cent, have an attack with arthritic symptoms, and one-sixth, or 16.8 per cent, an attack with carditis alone.

Recurrence in relation to age at onset.

Dividing the cases according to the age at onset and combining them into age groups of 3 years, it will be found (table 29) that the percentage incidence of children having one or more recurrences declines as the age at onset of the disease increases. This decline is fairly uniform for the first four age-groups but from the age of 13 years the risk of a recurrence seems to decline more markedly. (The number of cases with recurrence expressed as a percentage of the total number of patients in each age-group is: for 12—13 years of age, 45.8 per cent; 13—14 years, 28.6 per cent; 14—15 years, 35.1 per cent; and 15—16 years, 22.2 per cent.)

The percentage figure for cases with one or several recurrences is 56.8 for the ages 1—6 years, while the corresponding figure for the ages 13—15 years is 29.2. Thus, the recurrence rate

Table 29.

Recurrence rate in various age-groups.

Relation between the number of cases with recurrences and the total number of rheumatic fever cases in the four age-groups tested with the χ^2-method for heterogeneity.

Age-groups, in years	No. of cases surviving initial attack	No. of cases amongsurvivors where at least one recurrence occurred	%
1— 3 } 1—6	21 } 118	11 } 67	52.4 } 56.8
4— 6 }	97 }	56 }	57.7 }
7— 9	215	111	51.6
10—12	179	84	46.9
13—15	106	31	29.2
Total	618	293	47.4
		$\chi^2 = 19.8$, $P < 0.001$, d. f. 3	

in children whose initial attack occurs before school age is about double the rate for children whose initial attack occurs towards the end of childhood.

Recurrence rate.

Methods.

As the duration of the disease varies so greatly (from about 3 weeks up to 2—3 years for a single attack), the interval between two attacks has in this investigation been reckoned from the date at which the patient in question was regarded as cured from the last attack, to the onset of the fresh attack.

In order to calculate the risk of recurrence, the material was divided, from the standpoint of the time of the initial attack, into the two previously mentioned year-groups corresponding to the periods 1922—1931 and 1932—1940. Besides this, special

calculations were made with regard to the risk of recurrence in cases with chorea as the primary manifestation and in cases with the other primary manifestations of rheumatic fever. The risk of recurrence was also calculated for the two first half-years after the recovery from the initial attack, as well as for each whole year in the subsequent course. For these calculations and for the tabular summary the following symbols and formulae have been used:

A: Total number of cases observed for at least t years after the previous attack.

B: Number of cases with a recurrence during the year from t to $t+1$ after the previous attack.[1]

C: Number of cases that die from another cause than rheumatic fever or its sequelae during the year from t to $t+1$ after the previous attack.

D: Remaining cases that have, certainly, been observed for t, but not for $t+1$ years after the previous attack.[1]

E: $A - \frac{D}{2}$

F: $100 \times \frac{B}{E}$

G: $100 - \frac{B+C}{E} \times 100$

H: $\frac{G}{100} \times$ H in the previous row.

I: $\frac{F}{100} \times$ H in the previous row.

J: the sum of the I-values up to and including the respective period.

K: $100 - I - J$.

These letters thus have the following significance:

A, B, C, D: *the material.*

E, G, I: auxiliary numbers.

[1] In tables 30, 31, and 32 this period of time refers to half a year only in each of the first two rows.

F: *The risk of a recurrence between the points of time t and t+1 after the previous attack for those who are alive at the date t without having had the respective recurrence* (e. g. in table 30, of those who had lived for three years without having had a recurrence, 4.8 per cent had one during the fourth year).

From H, J (and K) *one finds how a group of, from the outset, 100 persons would have developed if it had remained intact and assuming the conditions otherwise to be those observed.*

H: *Number of survivors without recurrence at the end of the respective period.*

J: *Number of survivors that had a recurrence up to the end of the respective period.*

K: Number of cases that died from some other cause than rheumatic fever or its sequelae up to the end of the respective period.

In addition to the basic material (cols. A, B, C and D) tables 30—36 include only the calculations indicated in the above-mentioned items F, H and J.

Results.

The classification of the material into cases with chorea as the initial manifestation of rheumatic fever and cases with other primary manifestations, that was in part carried out in connection with the calculations, has not been shown here in tabular form; instead of this the result has been summarized in figure 13. As will be seen, there seems to be a tendency to an earlier appearance of recurrences in the group in which chorea was the initial manifestation, as compared with the group comprising other initial manifestations of rheumatic fever. This difference, however, gradually grows less, and when 8 years have elapsed after the end of the initial attack the percentage of cases that have had their first recurrence is about the same in each group. This circumstance seems to be in good agreement with the clinical experiences, according to which it would seem as if chorea shows a specially strong tendency to recurrence during the period

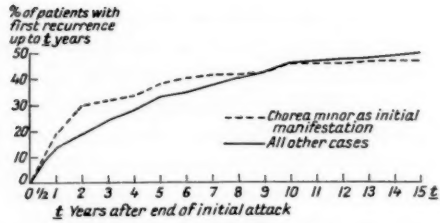

% of patients with first recurrence up to t years
50
40
30
20
10
0
Chorea minor as initial manifestation
All other cases
0 ½ 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 t
t Years after end of initial attack

Fig. 13. Rate for first recurrence up to end of respective t years after initial attack.

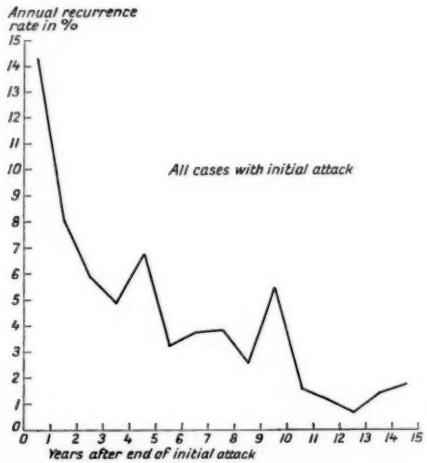


Fig. 14. Recurrence rate in various years after end of initial attack.

immediately subsequent to the initial attack. The fact that the two groups in the long run approach the same level, is perhaps, however, more remarkable. Considering only the first two years after the conclusion of the initial attack, the difference in the recurrence rate that has here been shown to exist between the two groups is significant (standard error calculated according to the formula in table 37).

There is thus not any considerable difference between the risk of recurrence (apart from that just mentioned) in the cases

Table 30.

Recurrence rate for first recurrent attack in those cases where the initial attack occurred in the period 1922—1940, calculated on the basis of the number of cases surviving the initial attack.

For explanation of lettering, see text on page 118.

Point of time t	A	B	C	D	F	H	J
0	589	51	—	—	8.7	91.3	8.7
½	538	34	1	—	6.3	85.4	14.4
1	503	40	2	—	8.0	78.3	21.2
2	461	27	—	—	5.9	73.7	25.8
3	434	21	1	—	4.8	69.9	29.4
4	412	27	—	20	6.7	65.8	34.1
5	365	11	—	43	3.2	63.2	36.2
6	311	11	1	29	3.7	60.6	38.5
7	270	10	1	11	3.8	58.1	40.8
8	248	6	—	15	2.5	56.6	42.2
9	227	12	—	10	5.4	53.6	45.3
10	205	3	—	19	1.5	52.8	46.1
11	183	2	—	18	1.1	52.1	46.7
12	163	1	3	13	0.6	50.8	47.1
13	146	2	—	12	1.4	50.1	47.8
14	132	2	—	23	1.7	49.3	48.6

with chorea[1] as the initial manifestation and in those whose primary manifestation of rheumatic fever consisted of arthritic symptoms[1] or carditis alone. These two groups have therefore been considered together in the rest of this chapter.

If one considers the total number of cases of rheumatic fever during the years 1922—1940 (table 30), one finds that the risk of a recurrence (col. F) declines with the number of years that elapse from the conclusion of the initial attack. The risk of recurrence during the first year after the initial attack is thus 14.4 %

[1] With or without carditis.

Table 31.

Recurrence rate for first recurrent attack in those cases where the initial attack occurred in the period 1922—1931, calculated on the basis of the number of cases surviving the initial attack.

For explanation of lettering, see text on page 118.

Point of time t	A	B	C	D	F	H	J
0	299	22	—	—	7.4	92.6	7.4
½	277	19	—	—	6.9	86.3	13.7
1	258	23	1	—	8.9	78.3	21.4
2	234	13	—	—	5.6	73.9	25.8
3	221	11	1	—	5.0	69.9	29.4
4	209	19	—	—	9.0	63.5	35.8
5	190	6	—	—	3.2	61.5	37.8
6	184	8	—	—	4.3	58.9	40.5
7	176	8	—	—	4.5	56.2	43.2
8	168	3	—	—	1.8	55.2	44.2
9	165	10	—	—	6.1	51.8	47.5
10	155	3	—	—	1.9	50.8	48.5
11	152	2	—	—	1.3	50.2	49.2
12	150	1	3	—	0.7	48.8	49.5
13	146	2	—	12	1.4	48.1	50.2
14	132	2	—	23	1.7	47.3	51.0

±1.5, but it sinks thereafter successively, and after 5 years it is about 3—4 per cent, decreasing to 1—2 per cent after the lapse of 10 years from the conclusion of the initial attack. This declining recurrence rate with an increasing number of years after the conclusion of the initial attack appears perhaps still more clearly from the diagram given in figure 14. As may be seen from tables 31 and 32, there is no considerable difference between the series in the two year-groups 1922—1931 and 1932—1940.

The total rate of recurrence (col. J, table 30) for the first 5 years after the initial attack is 34.1 per cent, for the first 10

Table 32.

Recurrence rate for first recurrent attack in those cases where the initial attack occurred in the period 1932—1940, calculated on the basis of the number of cases surviving the initial attack.

For explanation of lettering, see text on page 118.

Point of time *t*	A	B	C	D	F	H	J
0	290	29	—	—	10.0	90.0	10.0
½	261	15	1	—	5.7	84.5	15.2
1	245	17	1	—	6.9	78.3	21.0
2	227	14	—	—	6.2	73.4	25.9
3	213	10	—	—	4.7	70.0	29.8
4	203	8	—	20	4.1	67.1	32.2
5	175	5	—	43	3.3	64 9	34.4
6	127	3	1	29	2.7	62.6	36.1
7	94	2	1	11	2.3	60.5	37.5
8	80	3	—	15	4.1	58 0	40.0
9	62	2	—	10	3.5	55.9	42.1
10	50	—	—	19	0	55.9	42.1
11	31	—	—	18	0	55.9	42.1
12	13	—	—	13	0	55.9	42.1

years 45.3 per cent, while by the fifteenth year after the conclusion of the initial attack it has reached the figure 48.6 per cent. It may be said, in other words, that practically all the first recurrences have occurred within 10 years after the conclusion of the initial attack, and that after this lapse of time only very few cases have a first recurrence of rheumatic fever.

Is there any change in the recurrence rate in this material?

In chapter 3, it was shown that the tendency to a first recurrence seems to have diminished somewhat on a comparison between the cases with the initial attack in the earlier and those in the later part of this material, though in that connection regard was

Table 33.

Recurrence rate for second recurrent attack in those cases where the initial attack occurred in the period 1922—1931, calculated on the basis of the number of cases surviving the first recurrent attack.

For explanation of lettering, see text on page 118.

Point of time *t*	A	B	C	D	F	H	J
0	131	15	1	—	11.5	87.8	11.5
1	115	7	—	2	6.1	82.4	16.8
2	106	7	—	2	6.7	76.9	22.3
3	97	9	—	2	9.4	69.7	29.5
4	86	9	—	1	10.5	62.4	36.9
5	76	3	—	2	4.0	59.9	39.4
6	71	3	—	1	4.3	57.3	41.9
7	67	—	—	4	0	57.3	41.9
8	63	2	—	3	3.3	55.5	43.8
9	58	—	—	4	0	55.5	43.8
10	54	—	—	8	0	55.5	43.8
11	46	1	—	3	2.2	54.2	45.0
12	42	—	—	6	0	54.2	45.0
13	36	1	—	5	3.0	52.6	46.7
14	30	—	—	8	0	52.6	46.7

only paid to the first recurrence after the lapse of 4 and 7 years respectively from the end of the initial attack. This decline, however, was not sufficiently great to be described as statistically probable.

In the calculation of the recurrence rates for the total material a further comparison in this respect will therefore be made. For this purpose the material has, as already mentioned, been classified into two equal groups, containing patients having their initial attack in the years 1922—1931 and 1932—1940 respectively, and in which also the cases with chorea as the initial manifestation of rheumatic fever have been included. For these two year-groups tables have been drawn up to show the rates for the

Table 34.

Recurrence rate for second recurrent attack in those cases where the initial attack occurred in the period 1932—1940, calculated on the basis of the number of cases surviving the first recurrent attack.

For explanation of lettering, see text on page 118.

Point of time t	A	B	C	D	F	H	J
0	104	8	—	5	7.9	92.1	7.9
1	91	8	—	7	9.1	83.7	16.3
2	76	3	—	6	4.1	80.3	19.7
3	67	2	—	7	3.1	77.7	22.3
4	58	2	—	11	3.8	74.8	25.2
5	45	3	—	12	7.7	69.0	31.0
6	30	1	—	4	3.6	66.6	33.4
7	25	—	—	4	0	66.6	33.4
8	21	—	—	5	0	66.6	33.4
9	16	—	—	4	0	66.6	33.4
10	12	—	—	8	0	66.6	33.4
11	4	—	—	—	0	66.6	33.4
12	4	1	—	3	40.0	39.9	60.1

first and second recurrence (tables 31—34). Corresponding calculations were also made for the third recurrence, though these have not here been shown in tabular form. Owing to the small number of cases with three and four recurrences, the year-groups 1922—1931 and 1932—1940 have been combined into one year-group, 1922—1940, in tables 35 and 36 relative to the rates for the third and fourth recurrences.

In comparisons between the two year-groups 1922—1931 and 1932—1940 it was thought advisable, in calculating the standard error for the recurrence rates, to do this only for certain points of time. It was therefore decided to make these calculations for the periods 5 and 10 years after the conclusion of the last attack. These calculations are shown in table 37, while in table 38 are given the differences between the year-groups as

Table 35.

Recurrence rate for third recurrent attack in those cases where the initial attack occurred in the period 1922—1940, calculated on the basis of the number of cases surviving the second recurrent attack.

For explanation of lettering, see text on page 118.

Point of time t	A	B	C	D	F	H	J
0	67	17	—	2	25.8	74.2	25.8
1	48	3	—	4	6.5	69.4	30.6
2	41	2	—	4	5.1	65.8	34.2
3	35	2	—	1	5 8	62 0	38.0
4	32	—	—	1	0	62.0	38.0
5	31	2	—	5	7.0	57.7	42.3

regards risk of recurrence, as well as the standard error of the differences.

From table 38 it emerges that as regards the risk of a first recurrence during the first 5 years after the initial attack, the decline (= the difference) in the recurrence rate in the two year-groups, 3.6 % $\pm$ 4.0, is rather slight. This difference in the risk of recurrence increases somewhat, however, if the observation period is prolonged to 10 years, being then 5.4 % $\pm$ 6.0, but at the same time also the standard error increases.

A comparison between the two year-groups as regards the risk of a second recurrence, for those who have already survived a first recurrence, reveals a greater difference, however, a difference which proves to be about equally great when calculated for the first 5 years from the conclusion of the previous attack (11.7 % $\pm$ 7.2) as when calculated for the first 10 years (10.3 % $\pm$ 12.5).

This decline in the recurrence rate is still more striking if one compares the two year-groups in respect of the risk of a third recurrence, the difference here being 16.0 % $\pm$ 15.0. Owing to the small number of cases that have survived a second recurrence

Table 36.

Recurrence rate for fourth recurrent attack in those cases where the initial attack occurred in the period 1922—1940, calculated on the basis of the number of cases surviving the third recurrent attack.

For explanation of lettering, see text on page 118.

Point of time t	A	B	C	D	F	H	J
0	26	12	—	2	48.0	52.0	48.0
1	12	1	—	—	8.3	47.7	52.3
2	11	1	—	2	10.0	42.9	57.1
3	8	1	—	1	13.3	37.2	62.8
4	6	—	—	—	0	37.2	62.8
5	6	—	—	1	0	37.2	62.8

and that thus come into question for a third, the standard error here is also greater, however.

If, then, as has been done above, one compares the cases that had their initial attack during the years 1922—1931 with the cases having their initial attack during the period 1932—1940, one finds a certain, though rather slight, decline in the risk of a first recurrence for the latter year-group. Making similar comparisons in respect of the risk of a second recurrence, this decline is found to be considerably greater, and this appears to apply in a still higher degree to the risk of a third recurrence. Owing to the relatively high standard errors, however, the various differences have not been established as statistically significant and are not even statistically probable.

There is, however, as has been shown, a progressive increase in the differences when the first, second and third recurrences respectively are compared. Thus, although none of the individual differences can be designated as significant, they nevertheless give, when taken together, a definite impression that a decline has really taken place in the recurrence rates for the second half of the material. If one weighs together these three differences

Table 37.

Recurrence rates. A survey.

Year-group	Recurrence no.	Time in years from preceding attack within which recurrence could occur	Recurrence rate %	Standard error
a 1922—1931	1	5	35.8	2.8
b 1922—1931	1	10	47.5	2.9
c 1932—1940	1	5	32.2	2.9
d 1932—1940	1	10	42.1	5.3
e 1922—1931	2	5	36.9	4.4
f 1922—1931	2	10	43.8	5.1
g 1932—1940	2	5	25.2	5.6
h 1932—1940	2	10	33.4	11.4
i 1922—1931	3	5	42.8	8.3
j 1932—1940	3	5	26.8	12.4
k 1922—1940	1	5	34.1	2.0
l 1922—1940	1	10	45.3	2.5
m 1922—1940	2	5	32.4	3.5
n 1922—1940	2	10	40.1	4.7
o 1922—1940	3	5	38.0	6.9
p 1922—1940	4	5	62.8	12.4

Formula for the calculation of the standard error of the recurrence rates in tables 37 and 38.

By p_t is meant the percentual risk for a recurrence within t years of the preceding attack (column J in tables 30—36). Its standard error, $\varepsilon(p_t)$, has been calculated according to the formula

$$\varepsilon(p_t) = \sqrt{\frac{p_t(100 - p_t)}{N - 1}}.$$

The figure N in the denominator was determined to be $\frac{100}{q_t} N_t$, where N_t is the number of cases still under observation after the passage of t years

Table 38.

Recurrence rates. Comparisons.

Comparisons between year-groups in table 37	Recurrence no.	Time in years from preceding attack within which recurrence could occur	Difference in recurrence rate %	Standard error
a—c	1	5	3.6	4.0
b—d	1	10	5.4	6.0
e—g	2	5	11.7	7.2
f—h	2	10	10.3	12.5
i—j	3	5	16.0	14.9

from the preceding attack and still without any recurrence, while q_t is the percentage of cases without a recurrence t years after the preceding attack (obtained from column H for the point of time $t-1$).

This relatively complicated method of calculation is necessary owing to the fact that some cases disappeared from the material during the observation period before t years had passed after the preceding attack (column D). If no cases had disappeared, the above formula would agree with the commonly employed formula for the standard error (i. e. N will be the first number in column A). On the other hand, if cases disappeared the formula is to be regarded as merely an approximation to the exact formula for the standard error, a calculation which would be extremely complicated. The approximation allows an extra safety margin in these calculations.

by a method described by Fisher in Statistical Methods (§ 21.1) a result that can be designated as statistically probable ($\chi^2 = 13.2$, $P < 0.05$, d. f. 6) will be obtained. This probability increases in value if one remembers that certain of the standard errors upon which the calculation is based have been determined with a certain margin of safety (see table 37).

In order to make still clearer this decline in the recurrence rate when compared in the two year-groups, I have constructed two diagrams (figs. 15 and 16), from which emerges above all the diminished risk of a second recurrence for cases having their initial attack during the period 1932—1940.

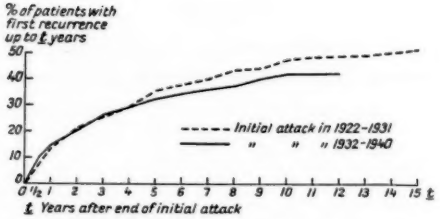

% of patients with first recurrence up to t years
50
40
30
20
10
0
Initial attack in 1922–1931
" " " 1932–1940
0 ½ 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 t
t Years after end of initial attack

Fig. 15. Rate for first recurrence up to end of respective t years after initial attack.

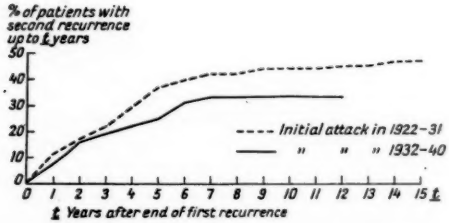

% of patients with second recurrence up to t years
50
40
30
20
10
0
Initial attack in 1922–31
" " " 1932–40
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 t
t Years after end of first recurrence

Fig. 16. Rate for second recurrence up to end of respective t years after first recurrence.

To the above calculations it may of course be objected that in the 1922—1931 year-group there may have been a larger number of patients, who were cared for at home during the whole of their initial attack, but who never had any recurrence and therefore were never brought to the notice of the hospital, while the contrary might conceivably be the case with the 1932—1940 year-group. The risk of a first recurrence would thus apparently be greater in the 1922—1931 year-group than in the 1932—1940 year-group, as the former group would not to the same extent include all initial attacks as would the latter group. This objection can, naturally, not be entirely dismissed. On the other hand, the risk of a second or third recurrence is not in the same way dependent upon the initial attack, as these recurrence calculations are based upon a known material, consisting of those patients who have already had their first recurrence. Moreover, these rates also showed a decline for the last-mentioned year-group,

a decline which even seems to be more pronounced than the decline in the risk of a first recurrence. These circumstances seem to me to indicate that the demonstrated decline in the recurrence rate really corresponds to the true conditions.

As regards the reason for the reduced recurrence rate thus demonstrated, the causes of the improved prognosis for the second half of the material have already been discussed in chapter 3, and what was said there probably applies, on the whole, also in this connection. It is, however, perhaps worth pointing out that, as a rule, all the patients from the 1932—1940 period, whether they were suffering from the initial attack or from a recurrence, were kept confined to their beds until the erythrocyte microsedimentation rate had gone down to the normal value, while for the first part of the material, containing patients having the initial attack during the period 1922—1931, the confinement to bed lasted as a rule only until other clinical signs of active infection had disappeared, which in other words meant a considerably shorter time under treatment. Moreover, as has already been mentioned, the control examinations have of late become increasingly thorough. It is very probable that these circumstances have in a high degree contributed towards causing the reduced recurrence rates, which in its turn must have played a part in lowering the death rate for those patients surviving the initial attack.

Comparisons between different attacks in respect of the risk of further recurrence.

If we compare the calculations in table 37, on the rates for the first, second, third and fourth recurrence respectively (= second, third, fourth and fifth attack), for patients already having survived an earlier (first, second or third) attack of rheumatic fever, we find that whatever the number of attacks survived, the recurrence rate is practically the same after each attack the patient passes through. The differences that can be shown are not even as great as their respective standard errors;

and this holds true whether one compares only the cases having their initial attack during the years 1922—1931 or 1932—1940 separately, or taken together, or whether one compares the differences during a five or ten-year period of observation.

In other words, this means that for an individual who has survived an initial attack of rheumatic fever or chorea, the risk of a first recurrence (= second attack) is the same as the risk of a second, third or fourth recurrence (= third, fourth or fifth attack) run by an individual who has survived his first, second or third recurrence.

Recurrence and mortality in relation to severity of initial carditis.

Using the principle of classification earlier applied to the cardiac affection during active rheumatic infection, according to which the cases were divided into "not definitely established,"[1] "slight," "moderate" and "severe" carditis, all the cases that died in connection with the initial attack will be referred to the group "severe carditis." It seems, however, to be of a certain interest to ascertain the fate of the patients surviving the initial attack, and to see whether, from the severity of the initial carditis, any information can be obtained regarding the outlook for the future.

In table 39, all the cases (with the exception of those with chorea as the initial manifestation) have been tabulated according to the severity of the initial carditis. As emerges from this, the risk of recurrence increases proportionally with increasing severity of the initial carditis, this increase, according to a χ^2-test, being statistically probable. As the carditis is, strictly speaking, an expression for the severity of the rheumatic infection, it can thus be said, in other words, that the severer the initial infection the more probable will it be that the patient will have one or more

[1] It should be noted that in the first half of the material electrocardiographic examinations were carried out in only a few cases in connection with the active infection.

Table 39.

Recurrence and mortality rates in relation to severity of initial carditis in all patients surviving initial attack. (Patients with chorea as the initial manifestation of rheumatic fever are not included.)

Relation between recurrence rate and severity of initial carditis in the various groups tested with the χ^2-method for heterogeneity. Relation between mortality due to or following recurrences and severity of initial carditis tested in the same way.

Severity of initial carditis [1]	Total cases [2]	Number of cases		Total deaths due to rheumatic fever [3]	Total deaths due to rheumatic fever [4]
		without recurrences	with recurrences		
Not definitely established	61 (+1)	40 65.6 % ± 6.1		—	—
			21 34.4 % ± 6.1	0	0
Mild	258 (+4)	164 63.6 % ± 3.0		—	—
			94 36.4 % ± 3.0	10 10.6 % ± 3.2	10 3.9 % ± 1.2
Moderate . .	69	40 58.0 % ± 5.9		—	—
			29 42.0 % ± 5.9	8 27.6 % ± 8.3	8 11.6 % ± 3.9
Severe . . .	41 (+1)	17 41.5 % ± 7.7		—	—
			24 58.5 % ± 7.7	14 58.3 % ± 10.1	14 34.1 % ± 7.4
			$\chi^2 = 8.1$, $P < 0.05$, d. f. 3	$\chi^2 = 26.3$, $P < 0.001$, d. f. 2	$\chi^2 = 41.7$, $P < 0.001$, d. f. 2

[1] For principles used for classification see text on page 80.

[2] Figures within brackets show number of patients who died of some other disease without having any recurrence.

[3] Percentage incidence in relation to all cases with recurrences in the respective groups.

[4] Percentage incidence in relation to the total cases in the respective groups.

recurrences. And, as also emerges from this table, the risk of a later fatal attack also increases proportionally with the severity of the initial attack, inasmuch as, of the initially severer cases, indicated as "moderate" and "severe" carditis, about one-fourth die in the course of some later attack. (A χ^2-test gives a rise that is statistically significant.) If one considers the mortality in later attacks in relation to the number of cases that had a recurrence, one finds that the mortality in the course of a later attack actually shows a still more marked rise, inasmuch as over 50 per cent (58.3 % $\pm$ 10.1) of the initially severe cases that later had a recurrence died in the course of one of these attacks.

On the other hand, of those patients who did not manifest unequivocal signs of carditis at the initial attack 34.4 per cent had, certainly, at least one recurrence, but not a single one of those who had a recurrence subsequently died.

Discussion.

As has already been pointed out in the chapter on earlier investigations, the recurrence rate mentioned by different authors shows considerable variation. The explanation of these divergences, as well as of the differences between other materials and my own findings is in all probability to be sought in different opinions as to what is to be regarded as a recurrence, differences in the length of the observation time, and differing conditions in the various geographical areas likely to affect the recurrence rate.

CHAPTER 6.

Prognosis as regards the heart.

Next in importance to the mortality in rheumatic fever is, perhaps, the question of whether permanent heart disease will arise or not. As has already been pointed out, the view that is becoming increasingly general is that practically no patient contracting rheumatic fever entirely escapes cardiac involvement in some form. All clinical experience shows, however, that this cardiac affection varies in the highest degree, as also that the signs of carditis present during the course of the disease have not infrequently practically disappeared by the time the disease itself has finally abated. The fact that the signs of heart damage remaining after recovery from rheumatic fever are often very slight or even absent does not, on the other hand, constitute a proof that the cardiac affection has passed off without causing permanent damage, since a follow-up examination after the lapse of one or a few years not infrequently shows that a valvular lesion has, in fact, developed. In general, one is also probably inclined to postpone the decision as to whether heart damage has developed or not until at least some years have elapsed since the abatement of the infection. The period of at least 4 years, and on average 12.2 years, that for all the cases in this material had elapsed from the initial attack of rheumatic fever until the follow-up examination was carried out should constitute a guarantee that the heart changes that may have been present had had time to become permanently established, and in any case could be taken as an expression for a persisting heart defect.

Nature and degree of cardiac affection from a comparison between patients with initial attack during the periods 1922—1931 and 1932—1940, respectively.

The already mentioned decline in the number of deaths during the period 1922—1940 seems to motivate an investigation to ascertain whether also the risk of contracting heart injury, as well as the nature and degree of this heart affection, has undergone any change in the one or the other direction. The observed decline in the number of fatal cases might conceivably be counterbalanced by an increase in the number of patients with severe heart deformity (the increase being caused by those patients who have passed through and survived severe carditis); on the other hand this increase in the number of patients with cardiac involvement might conceivably be compensated by a corresponding improvement in the prognosis for the remaining patients in the direction of a larger number of patients with no, or only very slight, cardiac affection.

In order to investigate this question, I tabulated all the patients according to the functional capacity of the heart[1] at the follow-up examination. The results may be seen in table 40, where all the patients with their initial attack falling within the periods 1922—1931 and 1932—1940 respectively, as well as the total material (also including the patients who had their initial attack before 1922), are shown in different rows. (The same system was adopted in tables 41 and 42, though these tables only comprise the surviving patients.)

[1] Functional classification according to "Nomenclature and Criteria for Diagnosis of Diseases of the Heart":

Class I. Patients with a cardiac disorder without limitation of physical activity. Ordinary physical activity causes no discomfort.

Class II. Patients with a cardiac disorder with slight to moderate limitation of physical activity. Ordinary physical activity causes discomfort.

Class III. Patients with a cardiac disorder with moderate to great limitation of physical activity. Less than ordinary physical activity causes discomfort.

Class IV. Patients with a cardiac disorder unable to carry on any physical activity without discomfort.

A study of table 40 indicates that the decline in mortality shown by a comparison of the year-groups 1922—1931 and 1932—1940 has not, as one might possibly have expected, resulted in an increase of the cases with severe cardiac involvement. Instead, one finds that it is the group best-situated from the functional viewpoint (Class I) whose percentage has increased in direct proportion to the decrease in mortality, while on the other hand, the group comprising cases without definite cardiac affection has practically the same percentage of cases in the two year-groups. The increase in the percentage incidence of cases in class I, from the 1922—1931 year-group to the 1932—1940 year-group, is, as a matter of fact, statistically significant (difference, 14.3 % $\pm$ 3.9). As appears from the table, however, there is also a certain increase in the cases belonging to class II. It should, however, be pointed out that the 1932—1940 year-group represents considerably younger ages than the 1922—1931 year-group, the number of years having elapsed between the initial attack and the follow-up examination being on an average 9.4 years more in the latter group than in the former. This circumstance may of course contribute to place the 1932—1940 year-group for the present in a more favourable position, not least in view of the fact that the heart has perhaps not to the same extent been exposed to the strain that the patients in the 1922—1931 group have had to stand; and this in its turn may perhaps mean that functional weaknesses have not yet been revealed.

If one studies in the two groups the number of patients with roentgenographic heart changes, one finds (table 40) that these also seem to show a similarly slight, though not so marked, increase. If, on the other hand, these cases be considered in relation to the number of cases that have survived their rheumatic infection (table 42), there proves to be no difference between the two year-groups.

A study of the electrocardiograms, on the other hand, shows that the number of patients with myocardial changes is equally large in each group.

It is probably also of interest to note the fact that if the number of patients without any definite, persisting heart changes (the group

Table 40.

Showing all patients grouped according to condition of heart at follow-up examination including those who had died before follow-up.

Years of onset	No. of cases with initial attack	No heart disease + possible heart disease	Functional classification			Deaths from		Roentgenographic heart changes
			Class I	Class II	Class III	Rheumatic fever	Other causes	
1922–1931	326	92 28.2 % ± 2.5	112 34.5 % ± 2.6	34 10.4 % ± 1.7	3 0.9 % ± 0.5	77 23.6 % ± 2.4	8 2.5 % ± 0.9	80 24.5 % ± 2.4
1932–1940	304	81 26.6 % ± 2.5	148 48.7 % ± 2.9	44 14.5 % ± 2.0	1 0.3 % ± 0.3	25 8.2 % ± 1.6	5 1.6 % ± 0.7	93 30.6 % ± 2.6
All cases	659	179 27.2 % ± 1.7	269 40.8 % ± 1.9	80 12.1 % ± 1.3	4 0.6 % ± 0.3	111 16.9 % ± 1.5	16 2.4 % ± 0.6	178 27.0 % ± 1.7

no heart disease + possible heart disease) be weighed against the total number of rheumatic fever cases in the two year-groups (table 40) no difference as regards percentage incidence between the two year-groups is observable, whereas on the other hand, such a difference is noted (table 41) if these cases are related to the number of survivors in the respective groups (the difference is 8.6 % ± 4.2).

Furthermore, it would seem to emerge from table 42 that it is above all the patients with mitral stenosis and the patients with both mitral stenosis and insufficiency that show an increase in the 1932—1940 year-group. And this circumstance may also be an indication of a less favourable prognosis as regards the heart in this year-group.

Finally, it should be pointed out that on the whole analogous conditions are found to exist if one makes separate investigations corresponding to those above, in patients manifesting at the initial attack, arthritic symptoms, carditis alone, or chorea.

Table 41.

Showing all cases grouped according to condition of heart at time of follow-up examination, in relation to total number of survivors.

Years of onset	No. of survivors	No heart disease	Possible heart disease	Functional classification		
				Class I	Class II	Class III
1922—1931	241	67 27.8 % ±2.9	25 10.4 % ±2.0	112 46.1 % ±3.2	34 14.1 % ±2.2	3 1.2 % ±0.7
1932—1940	274	47 17.2 % ±2.3	34 12.4 % ±2.0	148 54.0 % ±3.0	44 16.1 % ±2.2	1 0.4 % ±0.4
All cases	532	118 22.2 % ±1.8	61 11.5 % ±1.4	269 50.6 % ±2.2	80 15.0 % ±1.6	4 0.8 % ±0.4

Nature and degree of cardiac affection, and relation of cardiac affection to type of initial rheumatic manifestation.

To judge from the clinical picture, chorea seems seldom to be associated with pronounced carditis; rather the carditis is for the most part slight, if discoverable at all, during an attack of chorea. To some extent this also emerges from the already mentioned circumstance that only one case in this material died in connection with the initial attack of rheumatic fever when this took the form of chorea. It might therefore be assumed, on *a priori* grounds, that the patients having chorea as the initial manifestation would to a far lesser extent than other patients prove to have permanent cardiac disorder.

As clearly appears from tables 43, 44 and 45, however, this is by no means the case. A comparison between the patients with chorea initially and those with arthritic symptoms or carditis alone at onset shows practically no differences in respect of the

Table 42.

Showing all cases submitted to follow-up examination, grouped according to nature of heart lesion.

Years of onset	Total cases	No heart disease	Possible heart disease	MI	MS	MSI	ASI	A+M	VC+My	All roentgenographic heart changes	Roentgenographic heart changes but without sure enlargement of heart
1922—1931	241	67 27.8 % ±2.9	25 10.4 % ±2.0	82 34.0 % ±3.1	4 1.7 % ±0.8	30 12.5 % ±2.1	2 0.8 % ±0.6	21 8.7 % ±1.8	10 4.2 % ±1.3	80 33.2 % ±3.0	22 9.1 % ±1.9
1932—1940	274	47 17.2 % ±2.3	34 12.4 % ±2.0	102 37.3 % ±2.9	13 4.7 % ±1.3	51 18.6 % ±2.4	4 1.5 % ±0.7	15 5.5 % ±1.4	8 2.9 % ±1.0	93 34.0 % ±1.1	24 8.8 % ±1.7
All cases	532	118 22.0 % ±1.8	61 11.5 % ±1.4	188 35.3 % ±2.1	18 3.4 % ±0.8	86 16.2 % ±1.6	6 1.1 % ±0.5	37 7.0 % ±1.1	18 3.4 % ±0.8	178 33.5 % ±2.0	47 8.8 % ±1.2

MI: Mitral insufficiency.
MS: Mitral stenosis.
MSI: Mitral stenosis and insufficiency.
ASI: Aortic stenosis and insufficiency.
A+M: Aortic and mitral valvular affections present in conjunction.
VC+My: Vitium cordis of uncertain location + changes in the myocardium only (as proved by electrocardiograms).

degree and nature of the cardiac affection developing; and this remains true whether one studies the nature of the cardiac affection, the functional capacity of the heart, or the roentgenologic or electrocardiographic heart changes after the lapse of some years (on an average 12.2 years) from the initial attack. Instead, a strikingly good agreement in all respects is noted.

It will be shown later on (pp. 149 and 198) that this observation holds good, also, if one compares those patients in these two groups who never had a recurrence and those who, in every recurrence, manifested the same basic type (chorea or arthritic symptoms) of rheumatic infection. On the other hand, there are of course, as will also be shown, statistically very probable differences, as regards the existence of permanent heart disease, between patients who never had a recurrence and patients having one or more recurrences; there are also (see p. 144) such differences in respect of the severity of the carditis at the initial attack in patients with no recurrence.

If one studies tables 43 and 44 more closely, one finds that of those patients who survived their rheumatic attack about one-third (33.7 per cent) showed no definite signs of cardiac affection in the form of valvular deformity or myocardium damage. Of the total number of survivors, one-fifth (22.2 per cent), as a matter of fact, showed no signs whatsoever of any cardiac affection, while in one-tenth (11.5 per cent) of the cases the point is doubtful.

As regards the nature of the valvular deformity developing (table 43), the large majority of cases (about 62 per cent of the survivors) proved to have some form of mitral valve defect. The aorta alone was found to be involved in only 1 per cent of the cases, in all in 6[1] cases, aortic insufficiency alone being present in 4[2] cases, aortic stenosis in 1[3] case and aortic stenosis + insufficiency in 1 case, while a combined aortic + mitral defect was found in about 7 per cent of the cases.

---

[1] In 4 of these cases mitral involvement was also suspected.

[2] In 2 of these cases mitral involvement was also suspected.

[3] In this case mitral insufficiency was also suspected, a roentgenogram showing signs of mitral deformity.

Table 43.

Showing all cases examined at follow-up,

For abbreviations,

Nature of initial attack	Total cases	No heart disease	Possible heart disease	MI
Arthritic symptoms + carditis alone[1]	432	95 (1) 22.0 % ± 2.0	55 12.7 % ± 1.6	157 (8) 36.3 % ± 2.3
Chorea	100	23 23.0 % ± 4.2	6 6.0 % ± 2.4	31 31.0 % ± 4.6
All cases	532	118 22.2 % ± 1.8	61 11.5 % ± 1.4	188 35.3 % ± 2.1

As regards the state of the heart from the functional point of view (table 44), half of the survivors belonged to class I, and thus experienced no discomfort whatsoever from their cardiac disorder in connection with normal physical exertion. Only about 15 per cent of the survivors had heart deformity of such severity that ordinary physical exertion caused discomfort (Class II). Not quite 1 per cent were placed in class III.

Table 44.

Showing all cases examined at follow-up, grouped according to functional classification of heart.

Nature of initial attack	Total cases	No heart disease + possible heart disease	Functional classification		
			Class I	Class II	Class III
Arthritic symptoms + carditis alone[1]	432	150 (1) 34.7 % ± 2.3	216 (14) 50.0 % ± 2.4	63 (3) 14.6 % ± 1.7	3 (—) 0.7 % ± 0.4
Chorea	100	29 29.0 % ± 3.0	53 53.0 % ± 5.0	17 17.0 % ± 3.8	1 1.0 % ± 1.0
All cases	532	179 33.7 % ± 2.0	269 50.5 % ± 2.1	80 15.0 % ± 1.5	4 0.8 % ± 0.4

[1] Patients with carditis alone also shown within brackets.

43.

grouped according to nature of heart lesion.

see table 42.

MS	MSI	ASI	A+M	VC+My
13 (1) 3.0 %±0.8	60 (2) 13.9 %±1.7	4 0.9 %±0.5	31 (4) 7.2 %±1.2	17 (2) 3.9 %±0.9
5 5.0 %±2.0	26 26.0 %±4.4	2 2.0 %±1.4	6 6.0 %±2.4	1 1.0 %±1.0
18 3.4 %±0.8	86 16.2 %±1.6	6 1.1 %±0.5	37 6.9 %±1.1	18 3.4 %±0.8

Roentgenographic heart changes (table 45) occurred in about one-third of the surviving cases. About one-fourth of the total number of survivors (26.3 per cent) showed roentgenographic enlargement of the heart, while about 9 per cent only manifested changes indicating valvular disorder or the like, without any concomitant enlargement exceeding the ordinary normal values.

Signs of myocardial affection with or without concomitant valvular affection was found in 20.5 per cent of cases in this material.

Table 45.

Roentgenographic and electrocardiographic heart lesions.

Nature of initial attack	Total cases	No. of cases with roentgenographic enlargement of heart	No. of cases with roentgenographic changes but without cardiac enlargement	No. of cases with electrocardiographic changes
Arthritic symptoms +carditis alone .	432	107 24.8 %±2.1	36 8.3 %±1.3	88 20.4 %±1.9
Chorea	100	24 24.0 %±4.3	11 11.0 %±3.1	21 21.0 %±4.1
All cases	532	131 24.6 %±1.9	47 8.8 %±1.2	109 20.5 %±1.8

Table 46.

Cardiac affection at follow-up in relation to severity of initial carditis in all patients (incl

Relation between severity of initial carditis and that of final cardiac affec-tion. For principles used for classifi-catio

Nature of initial carditis	No. of cases with no recurrence	Nature of		
		No heart disease	Possible heart disease	No heart disease + possible heart disease
A. Not definitely established	59	19 32.3 % ± 6.4	14 23.7 % ± 5.5	33 55.9 % ± 6.5
B. Mild	189	48 25.4 % ± 3.2	24 12.7 % ± 2.4	72 38.1 % ± 3.5
Moderate	42	8 19.0 % ± 6.1	2 4.8 % ± 3.3	10 23.8 % ± 6.6
Severe	17	2 11.8 % ± 7.8	4 23.5 % ± 10.3	6 35.3 % ± 11.6
C. Moderate + severe . . .	59	10 16.9 % ± 4.9	6 10.7 % ± 3.9	16 27.1 % ± 5.8
Total	307			$\chi^2 = 10.6$, $P < 0.01$, d. f. 2

Cardiac affection at follow-up in relation to severity of initial carditis.

It is, of course, only natural that in the patients experiencing one or several recurrences the character of the recurrences will to a greater or lesser extent influence the heart and contribute to the possible development of heart disease. In order to be able to judge what the severity of the initial carditis has to tell us concerning the heart disease arising during the first attack, it therefore becomes necessary to study only those patients who never

46.

(incl. those with chorea as the initial manifestation) who never had a recurrence.

tion in groups A, B, and C tested with the χ^2-method for heterogeneity.
cation see text on page 80.

cardiac affection at follow-up

Functional classification			All cases with roentgenographic heart changes	Cases with roentgenographic heart changes but without definite enlargement of heart	Electrocardiographic heart changes
Class I	Class II	Class III			
25 42.4 % ± 6.4	1 1.7 % ± 1.7	—	9 15.3 % ± 4.7	3 5.1 % ± 2.8	6 10.2 % ± 3.9
100 52.9 % ± 3.6	17 9.0 % ± 2.1	—	51 27.0 % ± 3.2	22 11.6 % ± 2.3	31 16.4 % ± 2.7
23 54.8 % ± 7.7	9 21.4 % ± 6.3	—	19 45.2 % ± 7.7	5 11.9 % ± 5.0	8 19.1 % ± 6.1
8 47.1 % ± 12.1	3 17.6 % ± 9.2	—	4 23.5 % ± 10.3	1 5.9 % ± 5.7	5 29.4 % ± 11.1
31 52.5 % ± 6.5	12 20.3 % ± 5.2	—	23 39.0 % ± 6.4	6 10.2 % ± 3.9	13 22.0 % ± 5.4
	$\chi^2 = 12.0$, $P < 0.01$, d. f. 2		$\chi^2 = 8.4$, $0.01 < P < 0.02$, d. f. 2		$\chi^2 = 3.1$, $P > 0.20$, d. f. 2

had a recurrence. This has been done in table 46, where all cases have been divided up according to the functional capacity of the heart observed at the follow-up examination.

As might have been expected, the probability of developing permanent heart disease increases in proportion to the severity of the initial carditis, and at the same time the risk of contracting a severe heart lesion also increases. A χ^2-test shows, moreover, that this is statistically very probable.

Considering the heart from the roentgenologic viewpoint alone, the same tendency (table 46) to an increase in the number of cases with heart changes is observed. As cardiac enlargement or a

change in the heart silhuette is caused, at least in part, by the existence of a valvular deformity, this is of course only what might be expected.

A study of the electrocardiograms also shows a progressive increase in the percentage of cases with permanent myocardial changes in proportion to the severity of the initial infection (table 46), though this increase cannot be shown to be statistically probable.

It should be stressed as a noteworthy fact that even when no definite heart changes are detected during the rheumatic attack, a valve defect may nevertheless develop despite the fact that no recurrence has occurred. Of the 26 patients originally manifesting no certain carditis and found at the follow-up examination to have cardiac disorder, 1 had mitral stenosis, 18 mitral insufficiency, 4 mitral stenosis associated with mitral insufficiency, and 1 aortic insufficiency; in 2 cases an electrocardiogram revealed myocardium damage only. It should be pointed out that in this group with no definitely established carditis at the initial attack there are 21 patients who had chorea as the initial manifestation and of these, 10 proved to have heart disease at the follow-up examination (1 of these had mitral stenosis and 2 mitral stenosis in association with mitral insufficiency). — The explanation of the development of a heart lesion, without originally demonstrable carditis, may of course lie in subclinical rheumatic infections, but it may also lie in the inadequacy of our present diagnostic aids.

No less remarkable, it seems to me, is the circumstance that patients with originally severe carditis do not always develop permanent heart deformity. No fewer than 10 (16.9 per cent) of the cases originally referred to as having carditis of "severe" or "moderate" intensity proved at the follow-up examination to be free from all signs or symptoms of heart disease. (It is, of course, possible that further examinations, such as special tests for function, the hypoxemia load test, and so on, might in some cases have revealed slight cardiac changes, but the fact probably remains, nevertheless.) In a further 6 of these cases (10.2 per cent) the presence of cardiac disorder was doubtful. In other

words, one is not justified in drawing, from the severity of the original carditis, any definite conclusions as to whether the existing symptoms will disappear, though the probability that they will do so diminishes in proportion to the increasing severity of the carditis.

Cardiac affection at follow-up in relation to age at onset.

As has already been mentioned, the mortality rate declines as the age at the initial attack increases. The same relationship is

Table 47.

Showing all patients (including those who had died before follow-up) in age-groups of 3 years grouped according to condition of heart at follow-up examination.

Age at onset, in years	No. of cases	Nature of heart affection at follow-up			Death from		Roentgenographic heart changes
		No heart disease + Possible heart disease	Functional classification		Rheumatic fever	Other causes	
			Class I	Class II + Class III			
1— 3	27	7 25.9 % ±8.4	9 33.3 % ±9.1	2 7.4 % ±5.0	9 33.3 % ±9.1	—	7 25.9 % ±8.4
4— 6	106	24 22.6 % ±4.1	48 45.3 % ±4.8	8 7.5 % ±2.6	22 20.8 % ±3.9	4 3.8 % ±1.9	25 23.6 % ±4.1
7— 9	226	57 25.2 % ±2.9	93 41.2 % ±3.3	29 12.8 % ±2.2	40 17.7 % ±2.5	7 3.1 % ±1.2	62 27.4 % ±3.0
10—12	190	51 26.8 % ±3.2	76 40.0 % ±3.6	31 16.3 % ±2.7	31 16.3 % ±2.7	1 0.5 % ±0.5	54 28.4 % ±3.3
13—15	110	40 36.4 % ±4.6	43 39.1 % ±4.7	14 12.7 % ±3.2	9 8.2 % ±2.6	4 3.6 % ±1.8	30 27.3 % ±4.3
Total cases	659	179 27.2 % ±1.7	269 40.8 % ±1.9	84 12.7 % ±1.3	111 16.9 % ±1.5	16 2.4 % ±0.6	178 27.0 % ±1.7

apparent with regard to the recurrence rate. It therefore seemed of interest to investigate whether the cardiac affection observed at the follow-up had been influenced by the age at the initial attack.

Table 47 shows the whole series of cases worked up in the manner indicated. As will be observed, there is a slight increase in the percentage incidence of the more severe forms of heart lesion (Classes II and III) with increasing age, an increase which seems, in some measure at any rate, to be counterbalanced by the mentioned decline in the mortality. These changes are, however, not sufficiently pronounced to be statistically probable. If we consider the number of cases with roentgenographic heart changes we find as a matter of fact, that this number is of approximately the same magnitude no matter what the age was at the onset of infection.

Frequency of heart disorder and its nature in relation to occurrence or non-occurrence of recurrent attacks.

In a disease that, like rheumatic fever, attacks the heart, and moreover shows a fairly strong tendency to recur, there is an *a priori* possibility that the more attacks an individual passes through, the greater will be the probability that permanent heart disease will develop. In order to investigate this point, tables 48 and 49 were constructed, showing all cases in relation to the occurrence or non-occurrence of recurrent attacks, the cardiac lesions being distributed according to the functional capacity of the heart at the follow-up examination.

Comparing first of all the cases with chorea at the initial attack with all the other cases (table 48), it is found that in those without recurrence there is no undeniable cardiac involvement in proportionally the same number of cases in both the groups, this feature being apparent both when one considers the cases with definitely negative heart findings or those in which there is some doubt concerning the heart. If, on the other hand, one considers the number of cases with roentgenographic enlargement of the heart in the two groups without recurrence, this lesion seems to

occur in smaller numbers among the cases with initial chorea (the difference is not, however, statistically probable), while as regards persisting electrocardiographic heart changes no difference is observable.

Nor can one find here any particularly noticeable difference on comparing the cases with recurrence in the two groups (table 48), though this is not so surprising, since among the cases with chorea initially, also, there is a fairly high percentage of cases with other types of manifestation in the recurrent attacks.

If, on the other hand, one compares in each group (table 48) the cases without recurrence with those with one or more recurrences, one finds that, as might have been expected, the percentage incidence of cases manifesting no definite heart disease at the follow-up examination is higher among the cases without recurrence, the difference in the two groups being statistically probable. This decrease in the number of cases without established heart involvement in connection with the appearance of recurrent attacks causes an increase in the number of cases with heart deformity in the groups with the (from the functional point of view) severer heart lesions (Classes II and III), while the percentage incidence of cases with cardiac disorder causing no reduction in functional capacity (Class I) proves to be on the whole unchanged. This increase in the severer forms of heart lesion may perhaps be interpreted as indicating that in some cases, when recurrent attacks occur, lesions previously causing no limitation of physical activity become more severe, and this takes place in approximately the same proportions as it does in patients without definite heart lesions who develop manifest heart involvement. (The last mentioned cases, however, will, as a rule, belong to the group without reduction of functional capacity.)

The number of cases with roentgenographic heart changes shows in the two groups a statistically significant increase with the appearance of recurrent attacks.[1] The persisting myocardial

[1] For the group having chorea as the initial manifestation of rheumatic fever, however, this holds true only if one considers the cases with roentgenographic enlargement of the heart. For all roentgenographically demonstrable heart changes the difference in this group is 15.4 % ± 9.4.

Table 48.

Nature of cardiac affection at follow-up classified according to nature of initial

Initial manifestation		Survivors examined at follow-up	Nature of	
			No heart disease	Possible heart disease
Chorea	Without recurrences	51	15 (29.4 %)	5 (9.8 %)
			20	
			39.2 % ± 6.8	
	With recurrences	49	8 (16.3 %)	1 (2.0 %)
			9	
			18.4 % ± 5.5	
Arthritic symptoms with or without carditis + carditis alone	Without recurrences	256	61 (23.8 %)	40 (15.6 %)
			101	
			39.5 % ± 3.0	
	With recurrences	176	34 (19.3 %)	15 (8.5 %)
			49	
			27.9 % ± 3.4	

changes observed on the electrocardiograms are also found to have increased, but for the group with chorea initially the difference is not statistically probable, whereas on the other hand the difference for all the other cases is found to be very probable.

The observed increase in the number of heart defects, and the increase in the number of cases with severe cardiac lesions on the appearance of recurrent attacks of rheumatic fever as compared with the corresponding features in the number of cases without recurrence calls for more careful investigation into whether there is an increase in the severity of the heart disorder and whether the number of cases with heart lesions increases with an increased number of recurrences. Closer study of table 49 supports the assumption that this is the case. The number of cases with three or more recurrences is rather small, and I therefore preferred, for the purpose of the χ^2-test, to combine the cases with two or more recurrences into one group; this group was then compared with the cases with no recurrence and one only. Here, the percentage

48.

manifestation in relation to occurrence or non-occurrence of recurrent attacks.

heart affection			All cases with roentgenographic heart changes	Cases with definite roentgenographic enlargement of heart	Electrocardiographic changes
Functional classification					
Class I	Class II	Class III			
27 52.9 % ± 7.0	4 7.8 % ± 3 8	—	14 27.4 % ± 6.2	5 9.8 % ± 4.2	9 17.6 % ± 5.3
26 53.1 % ± 7.0	13 26.5 % ± 6.3	1 2.0 % ± 2.0	21 42.8 % ± 7.1	19 38.8 % ± 7.0	12 24.5 % ± 6.1
129 50.4 % ± 3.1	26 10.2 % ± 1.9	—	69 27.0 % ± 2.8	47 18.3 % ± 2.4	41 16.0 % ± 2.3
87 49.4 % ± 3.8	37 21.0 % ± 3.1	3 1.7 % ± 1.0	74 42.0 % ± 3.7	60 34.1 % ± 3.6	47 26.7 % ± 3.3

of cases with no definite heart disorder is seen to decrease with an increasing number of recurrences, and this is shown by χ^2-analysis to be statistically significant. The percentage of cases without limitation of physical activity (Class I) proves to be about the same when there are no recurrences as when there are one or more recurrences, while on the other hand the increase in the number of cases with cardiac lesions on the appearance of recurrent attacks occurs in the groups with severe cardiac disorder (Classes II and III), which increase is also statistically significant.

The number of cases with roentgenographic enlargement of the heart also shows the same increase with an increasing number of recurrences (the difference is statistically significant here also). The same applies to persisting myocardial changes observable on the electrocardiogram.

It is interesting to note that even in patients having several recurrences, permanent heart disease does not always develop. As emerges from table 49, the follow-up examination proved that

Table 49.

Nature of cardiac affection in relation to number of recurrent attacks in all cases examined at follow-up.

Relation between nature of cardiac affection and number of attacks in groups A, B, and F tested with the χ^2-method for heterogeneity.

Groups with the following number of recurrences	Survivors examined at follow-up	Nature of cardiac affection					All cases with roentgenographic heart changes	Cases with definite roentgenographic enlargement of heart	Electrocardiographic changes
		No heart disease	Possible heart disease	Functional classification					
				Class I	Class II	Class III			
A. 0	307	76	45	156	30	—	83	52	50
		121		50.8 % ± 2.9	9.8 % ± 7.1		27.0 % ± 2.4	16.9 % ± 2.1	16.3 % ± 2.1
		39.4 % ± 2.8							
B. 1	151	35	12	74	29	1	56	41	33
		47		49.0 % ± 4.1	19.2 % ± 3.1	0.7 % ± 0.7	37.1 % ± 3.9	27.2 % ± 3.6	21.9 % ± 3.4
		31.1 % ± 3.8							
C. 2	41	3	2	25	10	1	21	20	15
		5		61.0 % ± 7.6	24.4 % ± 6.7	2.4 % ± 2.4	51.2 % ± 7.8	48.8 % ± 7.8	36.6 % ± 7.5
		12.2 % ± 5.1							
D. 3	16	1	1	6	7	1	11	11	7
		2		37.5 % ± 12.1	43.8 % ± 12.4	6.3 % ± 6.1	68.8 % ± 11.6	68.8 % ± 11.6	43.8 % ± 12.4
		12.5 % ± 8.3							
E. ≥4	17	3	1	8	4	1	7	7	4
		4		47.1 % ± 12.1	23.5 % ± 10.3	5.9 % ± 5.7	41.2 % ± 11.9	41.2 % ± 11.9	23.5 % ± 10.3
		23.5 % ± 10.3							
F. ≥2	74	7	4	39	21	3	39	38	26
		11		52.7 % ± 7.0	28.4 % ± 5.2	4.1 % ± 2.3	52.7 % ± 5.8	51.4 % ± 5.8	35.1 % ± 5.6
		14.9 % ± 4.1							
		$\chi^2 = 16.7$, $P < 0.001$, d. f. 2			$\chi^2 = 25.6$, $P < 0.001$, d. f. 2		$\chi^2 = 18.9$, $P < 0.001$, d. f. 2	$\chi^2 = 38.8$, $P < 0.001$, d. f. 2	$\chi^2 = 13.2$, $P_{approx.} = 0.001$, d. f. 2

no fewer than 4 patients with more than two recurrences did not manifest any signs or symptoms of heart disease, and to these may be added a further 2 patients with "possible heart disease." It is of course possible that more thorough tests for function, and other heart-tests, might have succeeded in revealing slight myocardial damage, but this is by no means certain.

CHAPTER 7.

Electrocardiographic changes.

Some of the electrocardiograms made in connection with the follow-up examination showed certain changes, which will best be seen by studying table 50.[1] Pathologic changes of one kind or another were observed in the tracings of 109 out of 532 cases (20.5 per cent). In some of these cases, however, it was doubtful whether the changes were to be regarded as abnormal or not, since these cases only bordered on values generally considered to be pathologic, and since the age of maturity had not been reached by some of the patients in question. Excluding all cases where the electrocardiograms could be interpreted as not unequivocally abnormal, viz. 17 cases, 92 cases (17.3 per cent) were left.

In the case of patients with an abnormal P-wave only, 28 cases (22 certain and 6 uncertain cases, i. e. 25.6 per cent of 109 cases, and 5.3 per cent of 532 cases), it must, of course, be taken into account that this abnormality may have been due, at least in part, to the existing mitral valve lesion (or tricuspid valve lesion), and thus need not with absolute certainty constitute myocardial changes directly due to the rheumatic process. In all the patients with abnormal P waves, as a matter of fact, a valvular defect of the mentioned type was present, or, in a few cases, was strongly suspected. Assuming that this applies for all cases with an abnormal P-wave where no other change in the myocardium was simultaneously apparent in the tracing, and deducting them from the above-mentioned 109 and 92 cases with electrocardiographic abnormalities, there thus remain 81 (15.2 per cent) and

[1] In judging the electrocardiograms, the normal values given by Mannheimer (1940) and by Scherf and Boyd (1945) were used.

Table 50.

Electrocardiographic alterations observed at the follow-up examination.

Alterations	No. of cases
Abnormal P-wave	22
Abnormal P-wave?[1]	6
Abnormal P-wave + prolonged P—Q interval	7
Abnormal P-wave? + prolonged P—Q interval	1
Abnormal P-wave? + prolonged P—Q interval?	1
Abnormal P-wave + prolonged P—Q interval + abnormal QRS-complex	3
Abnormal P-wave + prolonged P—Q interval? + abnormal T-wave	1
Abnormal P-wave and QRS-complex	5
Abnormal P-wave + abnormal QRS-complex?	2
Abnormal P-wave and QRS-complex + abnormal S—T segment and T-wave	3
Abnormal P-wave? + abnormal QRS-complex + abnormal S—T segment?	1
Abnormal P-wave + abnormal S—T segment	2
Abnormal P-wave? + abnormal T-wave	1
Prolonged P—Q interval	8
Abnormal QRS-complex	10
Abnormal QRS-complex?	2
Prolonged P—Q interval + abnormal QRS-complex	1
Prolonged P—Q interval + abnormal QRS-complex?	1
Abnormal S—T segment and T-wave	7
Abnormal S—T segment	2
Abnormal T-wave	5
Abnormal S—T segment? + abnormal T-wave?	2
Abnormal S—T segment?	5
Abnormal T-wave?	1
Abnormal S—T segment and T-wave + prolonged P—Q interval	2
Abnormal S—T segment and T-wave + prolonged P—Q interval + abnormal QRS-complex	1
Abnormal S—T segment and T-wave + abnormal QRS-complex + prolonged P—Q interval?	1
Abnormal S—T segment or T-wave + abnormal QRS-complex	2
Abnormal S—T segment? + abnormal T-wave? + abnormal QRS-complex	1
Abnormal P-wave + prolonged P—Q interval + abnormal S—T segment and T-wave + abnormal QRS-complex + changes due to digitalis	1
Abnormal P-wave and QRS-complex + abnormal S—T segment and T-wave (levocardiogram and signs of coronary insufficiency)	1
Auricular premature systole with coupled beats + abnormal T-wave? and P-wave?	1
	Total: 109

[1] The question-marks denote borderline cases.

70 (13.1 per cent) cases with persisting disease of the myocardium following rheumatic infection. A point to be remembered here, however, is that causes other than a rheumatic infection may in some cases have underlain the electrocardiographic alterations, as, for instance, scarlatina, or diphtheria. It is less probable that the alterations could have been produced by changes due to age, incipient coronary sclerosis or other similar conditions, since the majority of the persons under examination were between the ages of 20—30 years, and only a few were as old as 30—35 years. The patients were not tested for syphilis by means of the Wassermann reaction in connection with the follow-up examination, but on the other hand this had been done in most instances when the patient was in the hospital, and in every case the result had been negative.

The total number of cases displaying changes in the P-wave amounted to 58 (47 certain and 11 uncertain cases), i. e. 10.8 per cent of 532 cases (or a percentage of 51.1 sure cases among the 92 cases with undeniable electrocardiographic alterations).

According to the literature, the commonest form of electrocardiographic alteration during rheumatic fever would seem to be a prolongation of the auriculo-ventricular conduction time or the P—Q interval. As may be seen from the table, there was persisting prolongation of the P—Q interval in 28 cases (5.3 per cent of the total 532 patients submitted to the follow-up examination); in 3 of these cases, however, this prolongation was just bordering on normal values. Studying more closely the remaining 25 cases (27.2 per cent of the 92 cases with definitely established electrocardiographic changes), the P—Q interval was found to be 0.24 sec. and 0.23 sec. respectively, in 2 cases (Record nos. 1022/1932 and 719/1934), while in the remaining cases the values were lower. Both these patients, who were born in 1922 and 1926 respectively, had had only one attack of rheumatic fever. The first patient had chorea, in association with mild carditis, the electrocardiogram showing, during the attack, P—Q values of 0.20—0.22 sec. and 0.16 sec., respectively, at two examinations. The second patient had arthritic symptoms as the initial manifestation, and in this case the P—Q interval at three examinations carried out at in-

tervals of up to 43 days was 0.20 sec., 0.21—0.22 sec., and 0.20 sec., respectively; in the tracing obtained at the follow-up there were also small changes in the QRS-complex.

In 4 of the cases with a prolonged P—Q interval the value was 0.21—0.22 sec., while in 7 cases it was 0.20—0.22 sec. To a certain extent, of course, these cases must be regarded as borderline cases, but they were counted as being pathologic, since the values exceeded 0.20 sec.

Of the patients with a prolonged P—Q interval, 12 were persons who had not yet reached the age of 18 years. Of these, 3 (of 12, 13, and 17 years of age), had a P—Q value of 0.21—0.22 sec. Six patients (of 14, 15, 15, 16, 17, and 17 years), had a P—Q interval of 0.20 or 0.20—0.21 sec. The remaining 3 cases (of 10, 11, and 12 years of age), had P—Q values of 0.18, 0.19, and 0.18 sec.

It should be stressed that none of these patients, as far as otherwise could be judged from the erythrocyte microsedimentation reaction and other tests, were in an active or subacute stage of the disease at the follow-up. One patient, however (a man of 21), with a P—Q interval of 0.21 sec., had had a mild attack (the fourth attack) a little over six months previously, for which he had been treated at home. Another patient, a 14 year old boy, had passed through a second attack about a year before the follow-up examination. None of the remaining patients had had an attack later than 4 years prior to the follow-up examination.

Changes in the QRS-complex were observed in 35 cases (30 certain and 5 uncertain cases, i. e. in 6.6 per cent of 532 cases, or 32.6 per cent[1] of the 92 sure cases). Alteration of the QRS-complex alone, without other electrocardiographic abnormality, was noted in 12 cases. Of these cases with an abnormal QRS-complex, a prolonged QRS-time ($>$ 0.10 sec.) was observed in 7 sure cases, while in a further 7 cases the prolongation was on the borderline of normal values (in these cases, however, there were other electrocardiographic abnormalities also, in the form of splitting or notching of the QRS-complex, which, considered together, caused a

[1] Calculated after exclusion of cases with electrocardiographic alterations of a doubtful nature.

diagnosis of disturbance of intraventricular conduction to be made).

There was very obvious notching or splitting of the QRS-complex (splintered initial deflections in lead III alone were not counted here) in 23 cases among the 35 with an abnormal initial deflection. Notching was only counted when it was present in more than one lead, and near the peak of the R-wave in one of the leads.

In 4 cases the initial deflection was of unusually high voltage, the R-wave in one or several leads exceeding 2.5 mv. In three of these four patients roentgenographic examination revealed enlargement of the heart. In 3 other cases the initial deflections showed an approximate value of about 2.5 mv., one of these patients proving also to have roentgenographic enlargement of the heart.

Abnormality of the S—T segment and T-wave was present in 40 cases (29 certain and 11 uncertain cases, i. e. in 7.5 per cent of 532 cases, or 31.5 per cent[1] of the 92 sure cases). An abnormal S—T segment (elevation above, or depression below, the normal limits), was noted in 7 cases, though 5 of these were borderline cases. An abnormal T-wave alone was observed in 6 cases (in one of these, however, the electrocardiogram was not undeniably pathologic).

As regards the alterations of the T-wave, these consisted in 14 cases of a wave of abnormally low voltage, complete absence, or a negative T-wave (changes of this kind in lead III were not taken into account). The T-wave greatly exceeded 0.5 mv. in 6 cases, while another 3 cases were interpreted as borderline cases.

Roentgenographic changes in conjunction with electrocardiographic alterations were found in 67 cases (61.5 per cent of the 109 cases with electrocardiographic changes). In 13 of these, however, there was only roentgenographic alteration of the heart silhuette without definite enlargement of the heart as judged by the principles already stated, while in the remaining 54 cases (49.5 per cent of 109 cases), there was roentgenographic enlargement of the heart in conjunction with electrocardiographic alterations.

---

[1] See footnote on page 157.

Among the afore-mentioned 13 cases there were 6 with electrocardiographic changes interpreted as not definitely abnormal. Three of these 6 cases showed alteration of the P-wave in their electrocardiographic tracing, and auscultation pointed to mitral insufficiency while the result of the roentgen examination made a mitral defect seem probable. Among the 54 cases with roentgenographic enlargement of the heart, 5 patients had only uncertain electrocardiographic abnormalities and 2 of these only had an abnormal P-wave (in these two cases the clinical and roentgenographic findings all pointed to a mitral valve defect). — If we now leave out of account those cases in which the electrocardiographic alterations were to be regarded as not definitely abnormal (11 cases), there remain 56 cases (10.5 per cent of 532 cases, and 60.8 per cent of the 92 cases with undeniable electrocardiographic abnormalities) that yielded both abnormal roentgenograms and electrocardiograms.

With regard to the cases with abnormal P-waves alone (28 cases), they all, with the exception of one (Record no. 687/1927), showed signs of mitral valve affection. In 4 cases, however, there was uncertainty on this point in connection with the physical examination, since the signs of valvular affection were not absolutely clear. In 2 of these 4 cases, however, roentgen examination yielded evidence favouring the diagnosis of mitral valve deformity. In the other 2 cases, in which the roentgenogram did not support a diagnosis of vitium cordis, the electrocardiographic abnormalities, as well as other signs and symptoms, made it possible to make the diagnosis of mitral valve involvement (?).

While no definite valvular defect could be found in 12 cases (although in most of them such disorder was more or less strongly suspected), the electrocardiograms nevertheless showed some abnormality.

One of these patients (Record no. 91/1926) had passed through three attacks with arthritic symptoms in association with undoubted carditis. On the electrocardiogram made at the follow-up there were, in addition to a number of changes of an uncertain nature (a slightly splintered or notched P-wave in leads I, II, and IV, voltage of T_I 0.5 mv. and of T_{II} 0.7 mv.) coupled beats of the auricular premature systole type. No

other signs of cardiac affection were obtained at the follow-up examination.

Another patient (Record no. 504/1922) had an attack of rheumatic fever with arthritic symptoms at the age of 9, and not quite one year later she had a second attack with arthritic symptoms and chorea, severe carditis with pericarditis developing during this attack. On her discharge from the hospital she showed signs of "insufficiency and stenosis of the mitral valve". At the follow-up examination, no signs of valvular affection could be detected and she had no subjective symptoms whatsoever. Electrocardiographic examination, however, showed a broad-topped, plump R_I of low voltage, notching of the initial deflection near the base line in lead III, and T-waves with a voltage not greater than 0.05 mv. in leads I and III and not greater than 0.1 mv. in lead II. The patient had gone through a partus without the least difficulty a few years before the follow-up examination.

Two patients (Record nos. 713/1925 and 758/1927) had had an attack with arthritic symptoms at 2 and 12 years of age, respectively, and a recurrence of the same type at 6 and 26 years, respectively. The former displayed at the follow-up slight disturbance of intraventricular conduction (with a QRS-time of 0.10 sec., a broad S_I and thickened R_{II}, and elevation of the S—T segment in lead IV), but these changes were not unequivocally pathologic. The other patient displayed slight and uncertain signs of myocardial damage in the form of an elevation of the S—T segments.

The other 8 patients without undeniable signs of valvular deformity had only had one attack of rheumatic fever.

Two of these 8 patients (Record nos. 840/1927 and 261/1934) had an abnormal QRS-complex alone, but in one, the alteration was to be regarded as bordering on normal. In both of these patients auscultation of the heart revealed a systolic murmur localized to the mitral valve, but the murmur was of such a nature that a sure diagnosis of valvular disorder could not be made. In a third case (Record no. 10/1937), a girl of 17 years old at the time of the follow-up, the P—Q time was 0.20—0.21 sec. but there were no other electrocardiographic abnormalities. She showed no signs of valvular deformity.

In two cases with no definite valvular disorder (Record nos. 1108/1924 and 904/1926) there was an abnormal initial complex and an abnormal S—T segment and T-wave. The latter patient had had an attack of rheumatic fever with arthritic symptoms and severe carditis in association with pericarditis at 9 years of age. No signs of valvular disorder were detected at the follow-up investigation. The former patient had had chorea minor without definitely established carditis at the age of 9, spending 68 days in hospital. At the follow-up examination, a systolic murmur was heard over the mitral valve, but this was not of

an unequivocally organic nature. Electrocardiographic examination revealed an abnormal S—T segment and T-wave, notching near the peak in the QRS-complex, and slightly plump S-waves in leads II and III.

In two other cases (Record nos. 176/1930 and 47/1939), both patients had had rheumatic fever with arthritic symptoms and a mild form of carditis at 7 and 13 years of age, respectively. An electrocardiographic examination carried out in connection with the follow-up showed abnormality of the S—T segment in one case, and an abnormal S—T segment and T-wave in the other.

The twelfth patient without definite valvular disorder (Record no. 687/1927), had had an attack of rheumatic fever with arthritic symptoms without definite carditis at 14 years of age. The follow-up examination revealed nothing to prove that he had valvular disorder, but his electrocardiographic tracing showed pointed P-waves in leads II and III. The P-wave in lead II had a voltage of 0.3 mv. and that in lead III of 0.25—0.28 mv. In this case it is not certain that these features on the electrocardiogram are to be interpreted as abnormal.

CHAPTER 8.

Various other signs and aspects and their prognostic significance.

Pericarditis.

Frequency.

Among the 689 rheumatic fever patients, there were signs of pericarditis at some stage of the disease in no fewer than 134 cases (19.4 per cent). Of these, 68 were boys and 66 girls. In 17 of these 134 cases, however, there was no mention of pericarditis in the case reports, the condition being first discovered in connection with the postmortem examination. But in several of these 17 cases the clinical picture was such that pericarditis might have been suspected even though it was not recorded in the case reports.

As regards the 117 cases (17.0 per cent of 689 cases) with clinically demonstrable pericarditis (see table 51), it was impossible to obtain any contact with two of them for the follow-up examination, the only information obtained being that both are still alive.

Of the patients with chorea as the initial manifestation of rheumatic fever, only 5 (3.9 % $\pm$ 1.7 of 127 cases) had clinically demonstrable pericarditis at some stage of the disease (3 of these cases had their pericarditis in connection with the initial attack). To these 5 cases must be added a further 4 where signs of an earlier pericarditis were not discovered until the postmortem examination (see table 51). Of these 9 cases (7.0 % $\pm$ 2.3), 8 died, while the survivor has not been available for a follow-up examination owing to emigration to America.

Because of the small occurrence of pericarditis among the cases with chorea as the initial manifestation of rheumatic fever

Table 51.

Nature of initial attack in relation to occurrence of pericarditis.

Nature of initial attack	Cases where the pericarditis was			Total cases
	Clinically observed	Not clinically observed	Observed clinically but patient not examined at follow-up	
Chorea	4	4	1	9
Arthritic symptoms . . .	83	11	1	95
Carditis alone	20	1	—	21
Arthritic symptoms + chorea later in same attack	7	1	—	8
Carditis alone + chorea later in same attack .	1	—	—	1
Total cases	115	17	2	134

these pericarditis cases have to a certain extent been treated as an independent group in this chapter.

Among the cases with carditis alone as the initial manifestation of rheumatic fever, on the other hand, the frequency of pericarditis (table 51) was very high, half of these cases (53.6 % ± 7.8) having on some occasion manifested signs of pericarditis. The high mortality already shown to occur among the carditis cases is probably very largely due to this circumstance.

Among the cases with arthritic symptoms at the initial attack, pericarditis was observed in 103 cases (19.8 % ± 1.8 of 521 cases).

As the borderline between the group with carditis alone and the group with arthritic symptoms is often extremely vague, especially in view of the fact that many of the cases had such very slight subjective symptoms from the joints and yet in spite of this have been included in the group with "arthritic symptoms," it seemed to me best to treat these two groups together in the following evaluations, though in certain connections, as already mentioned, I separated them from the group with chorea initially.

Table 52.

Showing patients who developed pericarditis (either during the initial attack or later), grouped according to age at their initial attack, in relation to total number of rheumatic fever cases with the initial attack at the respective ages.

Relation between cases developing pericarditis and total rheumatic fever cases in the age-groups of 3 years tested with the χ^2-method for heterogeneity.

Age at initial attack, in years	Total no. of rheumatic fever cases	Cases with pericarditis[1]	Cases with pericarditis in age-groups of 2 years[2]	Cases with pericarditis in age-groups of 3 years[2]
0— 1	—	—	—	
1— 2	1	—		
2— 3	12 27	2 (1)	8 (30.8 %)	8 (29.6 %)
3— 4	14	6 (2)		
4— 5	20	5 (1)	14 (24.6 %)	21 (18.9 %)
5— 6	37 111	9 (2)		
6— 7	54	7 (1)	20 (15.9 %)	
7— 8	72	13 (3)		46 (19.6 %)
8— 9	77 235	14 (1)	33 (20.2 %)	
9—10	86	19 (1)		
10—11	75	15 (—)	35 (23.8 %)	46 (22.8 %)
11—12	72 202	20 (2)		
12—13	55	11 (2)	19 (17.3 %)	
13—14	55	8 (1)		13 (11.4 %)
14—15	41 114	5 (—)	5 (8.5 %)	
15—16	18	—		
Total	689	134 (17)	134	134
				$\chi^2 = 7.9$, $0.05 < P < 0.10$, d. f. 4

[1] Cases where the pericarditis was not observed clinically also shown within brackets.

[2] The percentage incidence was calculated in relation to total number of cases in the respective age-groups.

Table 53.

Showing patients who developed pericarditis, grouped according to age at which this occurred, in relation to the number of patients who had their initial attack at the same age.

Age at initial attack, in years	Total rheumatic fever cases		Cases with pericarditis diagnosed clinically	Cases with pericarditis not diagnosed clinically	All known cases of peri-carditis	Cases with pericarditis in age-groups of 2 years[1]		Cases with pericarditis in age-groups of 3 years[1]	
0— 1	—		—	—	—	0			
1— 2	1		—	—	—				
2— 3	12	27	—	—	—	4	(15.4 %)	4	(14.8 %)
3— 4	14		2	2	4 (28.5 %)				
4— 5	20		3	2	5 (25.0 %)	11	(19.3 %)		
5— 6	37	111	5	1	6 (16.2 %)			19	(17.1 %)
6— 7	54		7	1	8 (14.8 %)				
7— 8	72		6	3	9 (12.5 %)	17	(13.3 %)		
8— 9	77	235	13	1	14 (18.2 %)			44	(18.7 %)
9—10	86		20	1	21 (24.4 %)	35	(21.5 %)		
10—11	75		14	—	14 (18.7 %)				
11—12	72	202	18	1	19 (26.4 %)	33	(22.4 %)	48	(23.8 %)
12—13	55		13	2	15 (27.1 %)				
13—14	55		10	2	12 (21.8 %)	27	(24.6 %)		
14—15	41	114	6	1	7 (17.1 %)			19	(16.7 %)
15—16	18		—	—	—	7	(11.9 %)		
Total	689		117	17	134	134		134	

Age at onset.

If in the patients that contracted pericarditis at some stage of the disease the age at which the initial attack of rheumatic fever took place is considered in relation to the total number of patients in the various ages (table 52), it will be found that the incidence of pericarditis appears to be highest when the initial

[1] The percentage incidence was calculated in relation to total number of cases in the respective age-groups.

Table

Comparison between year-groups 1922—1931 and 1932—1940 with respect initially

Years of onset	Total no. of cases with clinically diagnosed pericarditis	Total deaths among these[1]	Still living among these[1]	Nature of	
				No heart disease + possible heart disease	Functional Class I
1922—1931	58	40 69.0 % ± 6.1	18 31.0 % ± 6.1	4 6.9 % ± 3.3	8 13.8 % ± 4.5
1932—1940	49	20 40.8 % ± 7.0	29 59.2 % ± 7.0	4 8.2 % ± 3.9	20 40.8 % ± 7.0
All surviving patients grouped according to cardiac affection at follow-up: 49[4]				8 16.3 % ± 5.3	30 61.2 % ± 7.0

attack falls in the first 5—6 years of life (pericarditis is found in 25.6 per cent of the patients whose initial attack of rheumatic fever occurred before the 6th year of life). After this, the incidence of pericarditis remains at about the same level, 20 per cent, until the 13th year; from and including this year it sinks markedly. This decline, which according to the χ^2-analysis carried out may from the statistical point of view be due to chance, corresponds on the whole to the decline in the total mortality with increasing age that has already been mentioned (table 21, p. 102).

As in at least two-thirds (69.4 per cent) of all the patients pericarditis develops in connection with the initial attack (see table 56), and in 92.4 per cent this complication has already occurred by the end of the second attack, it is naturally to be expected that the age for contracting pericarditis will on the whole agree with

[1] Percentage incidence calculated in relation to total number of cases in the respective year-groups.

[2] Out of 70 known cases.

[3] Out of 50 known cases.

[4] Among 8 patients with their initial attack before 1922, who had pericarditis, 2 have survived and are included in this figure.

54.

to cases with their initial attack during these years and with pericarditis or later.

cardiac affection[1] classification		Roentgeno-graphic enlargement of heart[1]	Deaths within 4 years among cases with clinically diagnosed pericarditis[1]	Deaths within 4 years, among all pericarditis cases incl. those with pericarditis not diagnosed clinically
Class II	Class III			
5 8.6 % ± 3.7	1 1.7 % ± 1.7	8 13.8 % ± 4.5	30 51.7 % ± 6.6	42[2] 60.0 % ± 5.8
5 10.2 % ± 4.3	—	10 20.4 % ± 5.8	19 38.8 % ± 7.0	20[3] 40.0 % ± 7.0
10 20.4 % ± 5.8	1 2.0 % ± 2.0	18 36.7 % ± 6.9		

that for rheumatic fever in general, though of course slightly higher on the age-scale. This emerges fairly clearly, also, from table 53. As may be seen from this table, the peak incidence for pericarditis coincides with the peak incidence for rheumatic fever.

Is there any change in the prognosis for patients who contracted pericarditis similar to that described for the whole series?

As has previously been mentioned, a progressive improvement in the prognosis can be observed for the whole series on comparing patients contracting the disease in earlier years and those who had rheumatic fever in more recent years; this improvement is especially noticeable with regard to the mortality, but it is also to a certain extent apparent in the recurrence rate. Is it possible to trace similar tendencies among the patients contracting pericarditis?

In order to decide this question, I grouped the pericarditis material into patients having their initial attack during the years 1922—1931 and those contracting rheumatic fever in 1932—1940. For the sake of simplicity, and because on account of the

relatively small number of cases a distribution on smaller year-groups would have yielded uncertain statistical results, no further divisions were made (table 54).

As emerges from table 54, the number of patients who had a clinically established pericarditis is, certainly, smaller during the years 1932—1940 (15.8 per cent of 348 cases) than during the years 1922—1931 (16.7 per cent of 311 cases), but the difference is not very marked. The difference in the number of deaths (among cases contracting pericarditis) obtained between the two year-groups in respect of the death rate during the first four years after the initial attack (12.9 % $\pm$ 9.6) is, certainly, fairly large, but it is not even statistically probable. It gains in probability, however, when one remembers the improvement in the prognosis referred to earlier.

If, however, the cases dying during a 4-year period after the initial attack, and found at autopsy to have had pericarditis (table 54) are also included, then this difference rises (20.0 % $\pm$ 9.1), and now shows statistical probability. If the pericarditis group had been larger the statistical probability might, perhaps, have been greater.

It is remarkable that so many cases of pericarditis belonging to the 1922—1931 year-group (14 cases in all) were not discovered in connection with the clinical examination, whereas the corresponding number for the 1932—1940 year-group is only 1 case. A partial explanation of this is the lower mortality in the second half of the material, on account of which the cases with pericarditis not detected by a clinical examination have not yet come to our knowledge. On the other hand, the reduced mortality may perhaps be connected with a lowered incidence of pericarditis.

As regards the recurrence rate in the two groups, this can hardly be made the subject of a closer analysis, as both the total number of cases with pericarditis and above all the number of survivors after the initial attack are much too small.

It is also of interest to note here that the reduction in the mortality to be found in the 1932—1940 year-group as compared with the 1922—1931 year-group is counterbalanced by a striking increase in the incidence of heart disease in the group of what,

from the functional point of view, are the less severe cases (Class I), an observation which tallies with the conditions mentioned further back.

Mortality and recurrence rate.

Table 55 shows the cases of rheumatic fever (not including cases with chorea as the initial manifestation) divided up according to the occurrence of pericarditis in different attacks, as well as the mortality rate in the various attacks. Only cases with clinically demonstrable pericarditis have been included. In table 56 all cases of rheumatic fever (including those with chorea) and all cases of pericarditis (including cases not clinically observed) are assembled. As may be seen from these tables, the risk of contracting pericarditis diminishes as the number of attacks increases, this tendency bordering on statistical probability.

Of the patients who had pericarditis during the first attack of rheumatic fever and survived this (altogether 50 cases, though in 2 cases there is no information concerning possible recurrences), 25 (approx. 50 per cent) have had one or more recurrences. This is more or less the same as the recurrence rate obtained for the whole series of cases. Of the patients who did not have pericarditis until the second attack and who survived this (18 cases), 13 (72 per cent) have had at least one more recurrence.

Two patients who had signs and symptoms of pericarditis at the first attack manifested the same symptoms in the second attack also; and one patient who did not have pericarditis until the second attack manifested symptoms of pericarditis in the third attack also. The two first-mentioned patients are still alive, the last-mentioned patient died in connection with the third attack. Otherwise, no patients have shown symptoms of active pericarditis more than once.

The death rate with respect to persons contracting pericarditis in connection with rheumatic fever is, in this material, 61.9 per cent (table 56). The mortality seems to be about the same whether the pericarditis appears during the first attack or later. Removing all the cases in which chorea was the initial manifesta-

Table 55.

Frequency of pericarditis in various attacks in relation to all cases of rheumatic fever (not including cases with chorea as the initial manifestation), and frequency of patients who died after the respective attack or later.

Only cases with clinically detected pericarditis are included.
Relation between number of cases with pericarditis and total number of cases with the attacks indicated in the respective groups C, D, and E+F tested with the χ^2-method for heterogeneity.

	A No. of cases	B No. of cases with pericarditis (and percentage of A)	Total deaths among pericarditis cases (and percentage of B)	Deaths at attacks subsequent to pericarditis attack (and percentage of B)
C. Initial attack . .	549 [1]	78 [2] (14.2 % ± 1.5)	43 (55.1 % ± 5.6)	10 (12.8 % ± 3.8)
D. First recurrence .	230	27 [3] (11.7 % ± 2.2)	14 (51.8 % ± 9.6)	5 (18.5 % ± 7.5)
E. Second recurrence	74	4 [4] (5.4 % ± 2.6)	4 [4] (100.0 %)	—
F. Later recurrences	28	2 (7.1 % ± 4.5)	1 (50.0 %)	—
Total	549	111 (20.2 % ± 1.7) $\chi^2 = 5.6$, P approx. = = 0.05, d. f. 2	62 (55.8 % ± 4.7)	15 (13.5 % ± 3.2)

[1] 549 is the total number of cases where the number of recurrences is known. (The real total number is 562.)

[2] In 3 cases (not included) the initial manifestation was chorea; all three died, one at the initial attack. One case, with arthritic symptoms and pericarditis at the initial attack, and not examined at the follow-up, is not included here.

[3] In addition, there were 2 cases with pericarditis at the preceding attack also. (Both are living.) In 1 case (not included) the initial manifestation was chorea.

[4] In addition, there was 1 case with pericarditis at the preceding attack also. (This case died later.)

Table 56.

Table 56.

Frequency of pericarditis in various attacks in relation to all cases of rheumatic fever (including cases with chorea as the initial manifestation), and frequency of patients who died after the respective attack or later.

The table shows all cases of pericarditis, including those not detected clinically.
Relation between number of cases with pericarditis and total number of cases with the attacks indicated in the respective groups C, D, and E+F tested with the χ^2-method for heterogeneity.

	A No. of cases	B No. of cases with pericarditis (and percentage of A)	Total deaths among pericarditis cases (and percentage of B)	Deaths at attacks subsequent to pericarditis attack (and percentage of B)	Deaths at attacks subsequent to pericarditis attack. (Percentage incidence calculated on the basis of those patients who had further attacks of rheumatic fever.)
C. Initial attack . .	689	93 (13.5 % ± 1.3)	56 (60.3 % ± 5.1)	13 (14.0 % ± 3.6)	13 deaths among 25 patients with further recurrences (52.0 %)
D. First recurrence .	292	31 (10.6 % ± 1.8)	18 (58.0 % ± 8.9)	5 (16.1 % ± 6.6)	5 deaths among 13 patients with further recurrences (38.5 %)
E. Second recurrence .	98	8 (8.2 % ± 2.8)	8 (100.0 %)	—	
F. Later recurrences .	37	2 (5.4 % ± 3.7)	1 (50.0 %)	—	
Total	689	134 (19.4 % ± 1.5) $\chi^2 = 4.7$, $0.05 < P < 0.10$, d. f. 2	83 (61.9 % ± 4.2)	18 (13.4 % ± 2.9)	

Table 57.

Mortality in patients who had pericarditis in relation to number of attacks of rheumatic fever.

Pericarditis at:	Deaths at:	No. of cases detected clinically	No. of cases not detected clinically
First attack . . .	First attack	34	9
	Second attack	8	1
	Third attack	3	—
Second attack . .	Second attack	10	3
	Third attack	3	—
Third attack . . .	Third attack	4	4
Second, second, or fourth attack, respectively . .	Later than third attack	3	—
First attack . . .	Death from scarlatina 14 months after initial attack. (Pericarditis was conjointly present at initial attack)	1	—
	Total	66	17

tion of rheumatic fever, and at the same time considering only the pericarditis cases that were discovered at the bedside, the corresponding death rate will be 55.8 per cent (table 55).

As emerges from tables 55 and 56, the majority of deaths take place in connection with the attack during which pericarditis appeared, while only about 13—16 per cent of all the patients contracting pericarditis died in the course of a later attack. Of the 83 patients who died sooner or later after having had pericarditis, 64 (77.0 per cent) died at the attack during which pericarditis had appeared. (As regards the cases in which the pericarditis was not discovered until the postmortem examination, an assumption was made, on the basis of the clinical picture, concerning the attack at which the pericarditis appeared.)

Table 57 shows the mortality in relation to the number of attacks of rheumatic fever passed through.

Table 58.

Number of years from initial attack to death.

No. of years	No. of cases with clinically detected pericarditis	No. of cases with pericarditis not detected clinically	Total	% (of 83 cases)
$<\frac{1}{2}$	30	8	38	45.8
$\frac{1}{2}-1$	8	1	9	10.8
1—2	7	2	9	10.8
2—3	2	2	4	4.8
3—5	9	1	10	12.1
>5	10	3	13	15.7
Total	66	17	83	100.0

As regards the patients who had pericarditis and died 47 (56.6 per cent) died within one year of the initial attack of rheumatic fever, as may be seen from table 58. Within the course of 5 years 70 patients (84.3 per cent) had died.

Condition of heart at follow-up.

In table 54 is shown the functional capacity of the heart at the follow-up examination, in relation to the total number of rheumatic fever patients. In the bottom row in the same table is shown the functional capacity of the heart and the percentage incidence in relation to the number of survivors having had pericarditis at some stage of the disease. A comparison between this lower row and the findings in table 41, with respect to the whole material, does not reveal any striking differences except for the fact that the percentage of cases with no heart disease or possible heart disease taken together is much lower in the pericarditis group than in the whole material (the difference, 17.4 % $\pm$ 5.7, is statistically significant). Since pericarditis is an extremely grave complication following rheumatic fever, this circumstance seems to be quite natural.

Table 59.

Nature of cardiac affection in patients who were living after having had pericarditis.

For abbreviations see table 42.

No. of survivors	No heart disease	Possible heart disease	MI	MS	MSI	A+M	Electrocardiographic changes only
49	5 10.2 %	3 6.1 %	21 42.9 % ±7.1	1 2.0 % ±2.0	11 22.4 % ±6.0	6 12.2 % ±4.7	2 4.1 % ±2.8
	8 16.3 % ±5.3						

Considering the heart lesions from the standpoint of their location (tables 59 and 42), no particularly noticeable divergences from the average distribution for all cases is to be found.

An interesting feature is that there are some patients who, after having had severe rheumatic carditis in association with pericarditis, display no signs whatsoever of permanent cardiac lesions. In the 5 patients displaying no heart disease at the follow-up examination there had been no doubt whatsoever as to the correctness of the diagnosis of pericarditis.

Two of these patients (Record nos. 383/1923 and 344/1924) also had a recurrent attack at a later date, with arthritic symptoms. Both these patients are from the period when no electrocardiographic examinations were carried out. The other 3 patients had no recurrences (Record nos. 410/1923, 60/1926, and 529/1938). One patient (Record no. 410/1923) showed signs of failing cardiac function together with an affected general condition three weeks after the onset of rheumatic fever; this symptom subsided fairly rapidly, however. At the end of one month's illness he got a nosocomial diphtherial infection also. In the only patient (Record no. 529/1938) among those cases where electrocardiographic examination had been carried out during the acute rheumatic infection a heart block appeared, with every second beat not reaching the ventricles, during the most serious stage of his illness. An electrocardiogram recorded a few days later showed no pathologic features aside from a prolonged P—Q

interval of 0.33—0.35 sec. and possibly, also, a slightly prolonged Q—T time, but these abnormalities soon disappeared. She had a normal erythrocyte microsedimentation rate 45 days after the onset of the disease and was discharged from the hospital after a little over 80 days' illness.

In the 3 cases (Record nos. 10/1938, 156/1939, and 321/1939) where some uncertainty was felt at the follow-up examination as to whether heart damage was present or not, a soft systolic murmur was heard, with the point of greatest intensity over the cardiac apex, the organic nature of which was somewhat uncertain; otherwise, there were no signs of cardiac involvement. In one of these cases (Record no. 321/1939) the diagnosis of pericarditis must, however, be regarded as uncertain, seeing that the pericardial friction rub that was heard on one occasion only, on the fourteenth day after the patient's admission to the hospital, was not heard the next day. All of these 3 patients showed electrocardiographic alterations during the active infection; 2 of them showed prolongation of the P—Q interval, as well as other electrocardiographic signs of myocardial affection. One of these patients (Record no. 156/1939) had no demonstrable cardiac disorder, when discharged, either at the clinical or the electrocardiographic examination, despite the fact that, during the disease, he had had both systolic and diastolic murmurs located at the apex, as well as signs of conduction disturbances and other myocardial lesions.

In two cases (Record nos. 504/1922 and 904/1926) the follow-up examination revealed no signs of valvular affection, but on the other hand there were electrocardiographic alterations. (These cases are described on p. 160.)

The patient who, from the functional standpoint, was placed in class III (Record no. 1064/1929) had been taken ill at 9 years of age with a rheumatic infection in the form of mild carditis (only a harsh systolic murmur over the region of the cardiac apex, no electrocardiographic examination), and had received medical attention for about 8 weeks of 8 weeks. A little over two years later he had a fresh attack of rheumatic fever, pericarditis appearing after a little over 2 months in hospital. The erythrocyte microsedimentation rate did not go below 10 mm/hr, and remain there, until after about 490 days' illness. During the following winters, till he reached the age of 20, he had a recurrent attack, lasting up to a couple of months, almost every year, during which he had arthritic symptoms of varying intensity; for these attacks he usually received treatment at home. He had worked as a messenger, but at the time of the follow-up examination, when he was 24 years old, he was receiving an invalid pension. He was found to be suffering from a combined mitral and aortic lesion, with signs of both stenosis and insufficiency in both valves, fairly pronounced roentgenographic enlargement of the heart,

and electrocardiographic alterations indicative of grave myocardial damage. He seemed to be able to move about without much discomfort, but he became breathless on staircases and after slight exertion.

The cases placed in functional classes I and II showed nothing of sufficient interest to warrant special comment here.

The observations and findings resulting from the follow-up investigation indicated nothing to prove that the heart was adherent to the pericardium in any patient. Nor were there definite signs of fixation of the apex beat or of the right border of cardiac dulness, or of any systolic retraction in the region of the apex or diastolic shock over the precordium. No special electrocardiographic or roentgenographic examinations, or other special examinations for the purpose of ascertaining whether adhesion was present or not, were undertaken in connection with the follow-up investigation; I merely confined myself to ascertaining that the above-mentioned signs were absent. The roentgenograms revealed nothing to indicate calcification or other changes suggestive of a pericardial lesion, nor were there any signs of low voltage, or other such features, in the electrocardiograms. To make a diagnosis of pericardial adhesion is, as is well-known, so difficult that probably, before it could have been made, it would have been necessary, in the event of such a condition being suspected, to have the patient under observation at a hospital for some time. Not only would this measure undoubtedly have been impossible to carry out in most instances, but besides this, such an examination, although of much interest in this connection, must be regarded as not entirely relevant to the purpose of the present investigation.

Pleural affection.

That pleurisy, or a pleural effusion caused either by pleurisy or by stasis with a general tendency to edema, sometimes develop in connection with an attack of pericarditis is a well-known fact. In order to study this aspect in the present material I compiled table 60, from which it will be seen that pleurisy or a pleural effusion was present in 31 cases (4.5 per cent of the whole material).

Table 60.

Showing all cases with affection of pleura.

		No. of cases	Cases with concurrent signs of pericarditis	
			Observed clinically	Not observed clinically
Pleural effusion	Left	7	7	—
	Right	2	2	—
	Bilateral	7	3	3
	Total	16 [1]	12	3
Pleurisy	Left	8	7	1
	Right	3 [2]	2	1
	Bilateral	5 [3]	4	1
	Total	16 [3]	13	3
	Sum total	32 [1]	25	6

As may be seen, a right-sided affection alone was fairly uncommon. In all cases of pleural effusion except one, signs of pericarditis were concurrently present. In 6 cases, according to the case records, the findings from the pleura were the only clinical signs, although an autopsy proved later that pericarditis was also present.

That the pleurisy could have been of tuberculous origin in some of these cases may on the whole be regarded as unlikely, and nothing to prove that this might be the case was discovered. The presence of a positive tuberculin reaction in some cases is probably to be regarded more as a side issue.

[1] No definite signs of concurrent pericarditis in 1 case.

[2] Dry pleurisy in 1 case.

[3] Two attacks of pleurisy in 1 case, one during the initial attack and the other during the first recurrent attack. There were thus only 4 and 15 cases, respectively.

Table 61.

Incidence of pericarditis and mortality rate among cases with erythema annulare or nodules.

	No. of cases	No. of cases with pericarditis	Total no. of deaths
Erythema annulare . . .	40	7 17.5 % ± 6.0	11 27.5 % ± 7.0
Nodules	37	13 35.1 % ± 7.8	13 35.1 % ± 7.8
Erythema annulare or nodules or both symptoms	70	20 28.6 % ± 5.4	20 28.6 % ± 5.4

Subcutaneous nodules and erythema annulare.

In this material, subcutaneous nodules were present on some occasion in 37 cases (5.4 per cent of the total series of 689 cases), while erythema annulare occurred in 40 cases (5.8 per cent). In 7 of these cases the same individual had both erythema annulare and nodules at some time or other (at the initial attack or later).

As already mentioned, some authors consider the occurrence of nodules or erythema annulare to be a grave sign, since these lesions are only found in the severer cases. As will be seen (tables 61 and 62), my material shows no divergence from this conception.

Pericarditis was present in 19.4 per cent of the rheumatic fever cases. In the case of erythema annulare the frequency of this complication seemed to be about the same (17.5 per cent), while in the case of the subcutaneous nodule no less than 35.1 per cent showed signs of pericarditis at some attack. The difference between the whole series and this group with nodules (also included in the larger series) is statistically probable (15.7 % ± 6.3).

The mortality from rheumatic fever or its aftermaths is also much higher for patients with these symptoms than it is for the

Table 62.

Nature of cardiac affection at follow-up in cases with erythema annulare or nodules.

	No. of survivors examined at follow-up	Nature of cardiac affection			
		No heart disease	Possible heart disease	Functional classification	
				Class I	Class II	Class III
Erythema annulare	27 [1]	2	2	16	6	1
		4		59.3 % ± 9.5	22.2 % ± 8.0	3.7 % ± 3.6
		14.8 % ± 6.8				
Nodules	23 [1]	1	1	14	5	2
		2		60.8 % ± 10.2	21.7 % ± 8.6	8.7 % ± 5.9
		8.7 % ± 5.9				
All cases with nodules or erythema annulare	47 [1]	6		29	9	3
		12.8 % ± 4.9		61.7 % ± 7.1	19.1 % ± 5.7	6.4 % ± 3.6

whole series of cases. In the whole series, the death rate is 16.1 % ± 2.0, while for cases with erythema annulare it is 27.5 per cent (difference, 11.4 % ± 7.8), and for cases with nodules 35.1 per cent (difference, 19.0 % ± 8.2); thus here also, in the cases with nodules, a statistically probable difference. Taking the cases with these two symptoms together, the mortality is 28.6 per cent (difference, 12.5 % ± 5.8). In the 7 cases where both these two symptoms occurred in the same individual, the mortality rate is 56.0 per cent (4 deaths).

Turning to the condition of the heart found at the follow-up (table 62), the same tendency to a less favourable prognosis is found. Of the 4 cases which, from the standpoint of the functional capacity of the heart, belong to class III, no fewer than 3 had erythema annulare or subcutaneous nodules. If we glance at the number of cases with no definitely established heart deformity (the group with no heart disease + possible heart disease) it

[1] 1 case in each group died due to another cause than rheumatic fever. 1 case in the group with erythema annulare not examined at the follow-up.

will be found that the frequency of these cases is much lower among the cases with erythema annulare and subcutaneous nodules than it is in the whole series of cases (cf. table 41, p. 139). The difference is statistically probable (18.9 % ± 9.5) for cases with erythema annulare and very probable (25.0 % ± 10.3) for cases with nodules. Clubbing together all the cases with these symptoms the difference (20.9 % ± 7.0) is statistically significant.

As regards the risk of recurrence among the patients displaying these symptoms (because of the small number of patients surviving the initial attack the cases with nodules and erythema annulare have been clubbed together), 30 out of 54 patients (55.5 per cent) had one or more recurrences. From the statistical aspect, this figure is not very different from that applying to the whole series, although it is, of course, slightly higher for cases with these symptoms than it is for the other cases.

There thus seems to be little doubt, to judge from this material, that the appearance of erythema annulare or subcutaneous nodules in connection with rheumatic fever must be regarded as a bad omen, the occurrence of nodules being particularly unpropitious.

Microsedimentation rate of the red blood corpuscles.

As has already been mentioned, the erythrocyte microsedimentation rate was used chiefly in the latter half of the material as a guide to whether an active process was present or not. The group composed of patients who had their initial attack in the years 1922—1931 would therefore, on the whole, correspond to the patients treated till the fever and other clinical signs of infection had disappeared. The group comprising patients whose initial attack occurred in the years 1932—1940 consists, on the other hand, of those who were kept in bed, at the hospital, till the erythrocyte microsedimentation rate had gone down to 10 mm per hour and continued to remain at or below that level. In view of the course taken by rheumatic fever, this means a much longer

period in bed for patients in the second group as compared with those in the first.

In this connection it should be pointed out, however, that the microsedimentation rate of the red corpuscles usually remains at a very low level (as a rule, under 10 mm per hour) during the entire course, in patients with chorea as the initial manifestation of the disease. In such cases, of course, the disappearance of the choreic movements as well as of any other clinical signs or symptoms had to be used as the guide when deciding how soon the confinement to bed could be discontinued.

The improved, or gradually improving, prognosis established in several connections in the material under study makes it impossible to place the patients treated until the reinstatement of a normal erythrocyte microsedimentation rate in relation to those treated till other signs of the infection disappeared. Such a comparison yields, as already observed, a much better prognosis for the later group. It is, of course, conceivable, that the longer period in bed and the intensified after-care of the patients in the second group (with initial attack 1932—1940) may have contributed towards a lowering of the recurrence rate with the consequent lowering of the death rate, and also towards decreasing the risk that a permanent heart lesion will develop or that a lesion already in existence will become aggravated. This has, in fact, proved to be the case, but it must also be remembered that the same improvement in the prognosis may, alone or in part, be due to the previously mentioned possible change in the *genus epidemicus*.

The erythrocyte microsedimentation rate proved to be very high in all the patients (with the exception of the chorea minor cases). In most instances, the reaction was already at its maximum level when the patients presented themselves for medical treatment, although in a few cases, of course, it continued to increase during the next 2—3 weeks. In the vast majority of the cases (chorea cases not included) the rate exceeded 25 mm per hour but not 35 mm per hour, during the most acute stage of the initial attack, after which it gradually dropped, at varying rates

and with more or less marked undulations in some individuals, to below 10 mm per hour. Only in 24 patients was the rate over 35 mm per hour on one or several occasions, and in only 35 patients did it never exceed 25 mm per hour; in most of the last-mentioned cases, however, it was very close to this value. In only 1 case did the rate never go above 18 mm per hour.

This patient (Record no. 857/1930) had a recurrence within six year of onset, for which she was treated at home by a children's specialist. At the follow-up examination, in April 1944, she had no signs whatsoever of cardiac disorder.

From the prognostic standpoint, I have been unable to find any relation between the maximum level to which the erythrocyte microsedimentation rate rose and the prognosis for subsequent years, even though the question was examined from the aspect of the death rate, the recurrence rate, or the presence and severity of the cardiac lesion developing. In those patients having a recurrence at some later stage the risk of developing a permanent heart lesion increases, as has already been mentioned. Even when one only studies those patients where the erythrocyte microsedimentation rate never rose above 25 mm per hour and who never had a recurrence, no unequivocal difference as regards the prognosis can be observed, in this material, in comparison with those cases where the rate lay above that level.

If we examine, with regard to the patients surviving their initial attack of rheumatic fever (and excluding the chorea minor cases), how long a time elapsed from the onset of infection to the date when the erythrocyte microsedimentation rate returned permanently to a value of 10 mm per hour or under (tables 63 and 64), we find that there is nothing to prove that the prognosis might be different for patients who required varying lengths of time for a reinstatement of normal conditions.

However, the increase in the number of recurrence-free cases (table 63) among those cases where the treatment time had to be extended from 3 to 8 weeks before the erythrocyte microsedimentation rate returned to normal is certainly remarkable. In those cases where the sedimentation rate did not return to normal until after 8 weeks it was not uncommon

Table 63.

Recurrence rate in relation to number of days (during initial attack) required for a return to normal of the erythrocyte microsedimentation rate (M. S. R.).

Cases with chorea as the initial manifestation of rheumatic fever are not included.

Relation between number of cases with no recurrence and total number of cases with the respective treatment times shown in groups A, B, and C tested with the χ^2-method for heterogeneity.

M. S. R. < 10 after:	Total no. of cases	No. of cases with following no. of recurrences				
		0	1	2	3	≥4
A. ≤3 wks . . .	27	15 55.5 %	10 37.0 %	—	1 3.7 %	1 3.7 %
B. 4—5 wks . . .	58	38 65.5 %	14 24.1 %	4 6.9 %	1 1.7 %	1 1.7 %
C. 6—8 wks . . .	70	55 78.6 %	11 15.7 %	4 5.7 %	—	—
D. 9 wks—2 mths	64	45 70.3 %	15 23.4 %	4 6.3 %	—	—
E. 3—6 mths . .	41	26 63.4 %	11 26.8 %	2 4.9 %	2 4.9 %	—
F. > 6 mths . .	9	3 33.3 %	5 55.5 %	—	1 11.1 %	—
Total	269	182 67.7 %	66 24.5 %	14 5.2 %	5 1.9 %	2 0.7 %
		$\chi^2 = 5.6$, $P > 0.05$, d. f. 2				

to find that it had dropped at a slow rate, while in those cases where a normal rate was reinstated after a short illness the drop took place much more rapidly. The extension of the treatment time occasioned by the slow drop in the erythrocyte microsedimentation rate may conceivably have been an advantage, in that the longer and more effective hospital care involved a certain guarantee that a recurrence of the disease would not occur at a later date. A χ^2-test on the number of patients who had no recurrence in the groups with an erythrocyte microsedimentation rate of ≤ 10 after 3, 4—5, and 6—8 weeks of illness, respectively (see table

Table 64.

Cardiac affection at follow-up in relation to number of days (during initial attack) required for a return to normal of the erythrocyte microsedimentation rate (M. S. R.).

Cases with chorea as the initial manifestation of rheumatic fever are not included.

Relation between number of cases with no definite heart disease (= the groups »no heart disease» + »possible heart disease») and total number of cases with the respective treatment times shown in groups A, B, and C tested with the χ^2-method for heterogeneity.

M. S. R. < 10 after:	Survivors examined at follow-up	Cardiac affection					Roentgenographic enlargement of heart	Electrocardiographic changes
		No heart disease	Possible heart disease	Functional classification				
				Class I	Class II	Class III		
A. ≤3 wks	27	5 18.5 %	4 14.8 %	14 51.9 %	4 14.8 %	—	6 22.2 %	6 22.2 %
B. 4—5 wks	57	12 21.1 %	8 14.0 %	29 50.9 %	8 14.0 %	—	12 21.1 %	9 15.5 %
C. 6—8 wks	68	20 29.4 %	11 16.1 %	28 41.2 %	9 13.2 %	—	12 17.6 %	10 14.7 %
D. 9 wks—2 mths	61	12 19.7 %	6 9.8 %	36 59.0 %	7 11.5 %	—	15 24.6 %	8 13.1 %
E. 3—6 mths	39	3 7.7 %	5 12.8 %	22 56.4 %	8 20.5 %	1 2.6 %	10 25.6 %	11 28.2 %
F. >6 mths	9	—	—	7 77.8 %	2 22.2 %	—	6 66.7 %	2 22.2 %
Total	261	52 19.9 %	34 13.0 %	136 52.1 %	38 14.6 %	1 0.4 %	61 23.4 %	45 17.3 %
		$\chi^2 = 3.1$, $P < 0.20$, d. f. 2						

63) gave the result $\chi^2 = 5.64$, $P > 0.05$, d. f. 2. Thus, although the tendency is from the statistical standpoint almost probable ($P < 0.05$ requires a χ^2-value of 5.99) the figures obtained may nevertheless be due entirely to chance.

Another piece of evidence favouring the assumption that a longer period in bed during the initial attack brightens the prognosis, is the

circumstance (table 64) that, in the previously mentioned 3 groups, the percentage incidence of cases with no heart disorder or no undeniable heart disorder was found at the follow-up to increase with an increase of from 3 to 6—8 weeks in the length of the treatment time during the initial attack. Because of the small number of patients with no heart lesion, this increase is, however, not statistically probable.

Although the chance of escaping a permanent heart lesion is to some extent correlated with the chance of escaping a recurrence, these two observations may nevertheless be said to support one another, and thus in some measure to point to the important part played by an adequately long and effective confinement to bed in reducing the risk of later recurrences and increasing the chances of escaping permanent cardiac involvement, or at least of only sustaining mild heart damage.

In those cases where more than two months elapsed before the erythrocyte microsedimentation rate returned to normal it must be assumed that either the infection was of a severe nature right from the start or the individual powers of resistance were low, which in its turn must imply an increased danger that the disease will recur and also that it will more often cause permanent heart damage. This proved, also, to be the case, as may be seen from table 64.

In chorea minor, as mentioned before, the erythrocyte sedimentation reaction behaves in quite a different way than in other forms of rheumatic fever. By far the most usual occurrence, in chorea, is for the rate to remain at a level under 10 mm per hour during the whole of the chorea attack, possibly rising as the choreiform symptoms disappear, a feature that is especially noticeable when the symptoms of chorea minor, without any special interval with freedom from symptoms, pass over into arthritic symptoms. (The opposite occurrence, a fairly rapid fall in the sedimentation rate to less than 10 mm per hour, is often observed when an attack with arthritic symptoms or carditis alone changes over into chorea.)

It may often happen, that an individual can have a mild form of chorea minor for a fairly long time without any steps being taken to seek medical advice, mainly because the condition is not recognized as a disease but rather believed to be merely a nervous complaint. Because of this, the children often do not

come under medical treatment at an early stage of the disease, which in its turn means that it cannot be determined with certainty whether the erythrocyte microsedimentation rate was previously high and had then returned fairly rapidly to the normal value found at the first consultation. In the present series of chorea cases, however, very few of the patients came under treatment later than three weeks after the first appearance of the symptoms, at least as regards those who received hospital attention at all during their initial attack. If, therefore, we take it that, allowing for the difficulty of determining the exact date of onset, the erythrocyte microsedimentation rate did not, as a rule, change to any great extent during the first 2—3 weeks, and that, if the rate was low, it had in all probability been so from the inception of the disease, then 40 cases[1] in my material showed a rate of under 10 mm per hour during the first 2—3 weeks and for the rest of the attack, and 21 cases[1] a rate higher than 10 mm per hour but in most instances not higher than 15 or, at the most, 20 mm per hour (in one or two cases it was 25—30 mm per hour); in addition to this, there were 3 cases with a rate under 10 mm, but in these the reaction was not tested till after more than three weeks had elapsed since the onset of the infection. In the remaining 63 patients with chorea minor as the initial manifestation, the erythrocyte microsedimentation rate was not tested during the initial attack.

From the prognostic standpoint, there was no difference whatsoever between cases with a rate of under 10 mm during the whole course and cases where the rate was at a higher level on one or more occasions. Thus, although the number of cases was small (40 and 21 cases, respectively) there was no difference between the two groups in relation to the number of cases with, and those without recurrence, nor to the total number of deaths (one in each group), nor to the nature or functional degree of the cardiac affection found at the follow-up examination. The, on the whole, fairly small differences apparent between the two groups are of such a nature that they are undoubtedly to be ascribed to chance.

[1] 10 patients in each group had pharyngitis on their admission to the hospital.

Looking at the patients who, in connection with a recurrent attack of rheumatic fever, had chorea on some occasion, we find the same features as regards the erythrocyte microsedimentation reaction; in other words, the rate is, in most instances under 10 mm per hour. In patients who always had chorea both at the initial attack and at the recurrences, the reaction showed the same features. Thus, it was found, in three patients who had had more than one attack of chorea, that the rate was always low, under 10 mm per hour, while in two patients with a rate of over 10 mm at the initial attack it was under 10 mm at the recurrent attack.

Anemia.

When the criteria for the diagnosing of rheumatic fever were being discussed it was pointed out that according to some authors a more or less severe anemia is not infrequently present, a fact considered to be a useful aid for differential diagnosis when difficulty is experienced in distinguishing between pure pharyngitis in a rheumatic person and true rheumatic activity. Anemia in rheumatic fever was described as early as the eighteen-eighties, by Hayem, the French hematologist, who held that acute polyarthritis is the disease in connection with which the erythrocyte component of the blood suffers the greatest damage (Stransky, 1928). In Sweden, the same fact has been stressed especially by Gezelius.

It therefore seemed to be of interest to investigate the extent to which anemia was present in my material and what effect the existence of anemia might have on the prognosis. It should be mentioned first of all that more regular analyses of the blood were only carried out in the second half of the material, while in the first half the hemoglobin values were determined and an erythrocyte count made, as a rule, only in patients suspected of having anemia. Therefore, when studying whether anemia was present or not, I confined myself to cases where blood counts had been made with a fair degree of consistency, in other words, to patients whose initial attack occurred in the years 1930—1932 and up to

and including 1940. Further, for the sake of simplicity, I studied the hemoglobin value only, taking a value of ≥ 70 per cent (Autenrieth) as the normal level and counting all values below this as pathologic. In cases where there was only one value below 70 per cent and where the red blood cell count remained more or less at the same level as that found at preceding and subsequent examinations, I did not count this as pathologic. As in the case of the erythrocyte microsedimentation reaction, only the conditions at the initial attack were studied, since the conditions at this attack seem to me to be of most value in the judging of the prognosis.

Cases with arthritic symptoms or carditis alone at the initial attack.

In a publication on the blood changes in rheumatic diseases, Kahlmeter (1934) reported that anemia with erythrocyte counts of less than 4 million was present in 65 per cent of 200 patients with Bouillaud's disease. In half the cases this degree of anemia was already present one week after onset. As, in my material also, the anemia was present in most of the cases during the first few weeks, I decided to investigate what could be learned as to prognosis from an anemia developing during the first three weeks of the disease, the cases in question being compared with those having no anemia whatsoever in connection with their initial attack. To these were added those patients in whom anemia did not develop until after the lapse of three weeks from the onset of the disease.

The cases studied, which thus constitute all the cases where blood analyses were fairly consistently carried out, amounted to 280 (table 65). Of these, 105 cases (37.4 per cent) showed signs of anemia at some time during the course of the initial attack. In 15 cases (5.4 per cent) the anemia did not appear until after the lapse of three weeks from the inception of the disease, while in the remaining 32.0 per cent anemia developed in the course of the first three weeks.

It is, of course, undoubtedly so, that the more serious the rheu-

Table 65.

Mortality and incidence of pericarditis cases in relation to the hemoglobin value during the initial attack. (Cases with chorea as the initial manifestation of rheumatic fever are not included.)

Hb. value (Autenrieth)	Total cases	No. of deaths at initial attack	Total deaths	No. of cases with pericarditis at initial attack, and percentage of total cases	Mortality and mortality rate among pericarditis cases
Always ≥ 70 % . . .	175	2 1.1 % ± 0.8	4 2.3 % ± 1.1	14 8.0 % ± 2.1	4 28.6 % ± 12.1
Dropped during first three weeks to 60—69 %	67	7 10.4 % ± 3.7	9 13.4 % ± 4.2	16 23.8 % ± 5.2	8 50.0 % ± 12.5
Dropped during first three weeks to < 60 %	23	10 43.5 % ± 10.3	10 43.5 % ± 10.3	11 47.8 % ± 10.4	9 81.7 % ± 11.7
Dropped below 70 % for first time after lapse of first 3 weeks from onset of rheumatic infection	15	8 53.3 % ± 12.9	11 73.3 % ± 11.4	8 53.3 % ± 12.9	8 100.0 % ± 3.5

matic infection the greater is the possibility that anemia will be present. It is therefore of interest to find out whether, on the basis of the hemoglobin values obtained, any conclusions as to prognosis can be drawn, and whether there is any difference between milder forms of anemia and the severer forms. As may be seen from table 65, the mortality rises proportionally to a declining hemoglobin value (= the hemoglobin value during the first three weeks of the disease), this rise in the mortality proving by χ^2-analysis to be statistically significant.

Considering now the number of patients in the three groups with a severe form of rheumatic fever, which here may be said to be equivalent to the number of cases having pericarditis during the initial attack, we find that no less than one-third of the 41 cases with signs of pericarditis belong to the group with normal hemoglobin values during the whole of this attack. The percen-

Table 66.

Number of cases with and without recurrences in relation to the hemoglobin value during initial attack. (Cases with chorea as the initial manifestation of rheumatic fever are not included.)

Relation between number of cases with no recurrence and total number of survivors in groups I, II, and III tested with the χ^2-method for heterogeneity.

Hb. value (Autenrieth)	No. of cases surviving initial attack	No. of cases with no recurrence	No. of cases with one or more recurrences
I. Always ≥70 %	173	125 72.3 % ± 3.4	48 27.7 % ± 3.4
II. Dropped during first 3 weeks to 60—69 %	60	35 58.3 % ± 6.4	25 41.7 % ± 6.4
III. Dropped during first 3 weeks to <60 %	13	5 38.5 % ± 13.5	8 61.5 % ± 13.5
Dropped below 70 % for first time after lapse of first 3 weeks from onset of rheumatic infection .	7	2 28.6 % ± 17.1	5 71.4 % ± 17.1
		$\chi^2 = 9.0$, $P < 0.01$, d. f. 2	

tage incidence of pericarditis shows, naturally enough, a rise with decreasing hemoglobin values.[1] An interesting feature is that in pericarditis cases where the hemoglobin value does not drop below 70 per cent during the first three weeks of the rheumatic infection, the mortality rate is only 25—30 per cent, while in cases where the value drops to 60 per cent at the minimum during the same time, the death rate rises to 50 per cent, and in cases where the value drops below 60 per cent the mortality rate greatly exceeds 50 per cent.

The relatively small number of cases in the two anemia groups makes these figures a little uncertain, of course, but with this

[1] Actually, however, it is the anemia that develops, as a rule, in connection with a complicating pericarditis.

Table 67.

Cardiac affection at follow-up in relation to presence or absence of anemia at initial attack. (Cases with chorea as the initial manifestation of rheumatic fever are not included.)

Hb. value	No. of cases examined at follow-up	No heart disease + possible heart disease	Functional classification of cardiac affection		Roentgenographic heart changes	Electrocardiographic changes
			Class I	Classes II and III		
≥70	168	53 31.5 % ± 3.6	89 53.0 % ± 3.9	26 [1] 15.5 % ± 2.8	54 32.2 % ± 3.6	31 18.4 % ± 3.0
<70	70	24 34.3 % ± 5.7	34 48.6 % ± 6.0	12 [2] 17.1 % ± 4.5	28 40.0 % ± 5.8	19 27.2 % ± 5.3

reservation it could be said that when pericarditis appears during the opening course of a rheumatic infection a normal hemoglobin content during, as well as before, the appearance of this complication means a brighter outlook for the future than if anemia is present or develops.

Table 66 gives an idea of the extent to which the hemoglobin values at the initial attack can yield information regarding the risk of having a recurrence. As may be seen, the increase in the recurrence rate runs parallel with decreasing hemoglobin values at the initial attack, in those cases surviving this attack.

On the other hand, a low hemoglobin value during the opening attack seems to be of no great significance as far as the future cardiac affection is concerned (table 67).

If one studies the patients with a *recurrence* who were treated at the Children's Hospital for their recurrent attack, one finds approximately the same conditions with regard to the hemoglobin values as those described above, but because of the small number of cases in the various groups it did not seem worth while to work up any figures in tabular form.

[1] No case belonging to class III.

[2] Only 1 case belonging to class III.

Cases with chorea as the initial manifestation.

In no case where chorea was the initial manifestation of rheumatic fever was a low hemoglobin value observed. In the 41 cases where it was possible to study the hemoglobin value, 40 were found to have a value of over 70 per cent, 31 of these having a value over 75 per cent, while in one single case the values were 63, 74, and 76 per cent at tests made at one-week intervals. Not one of these patients has since died.

Tonsillectomy as a therapeutic measure in rheumatic fever.

The value of tonsillectomy for the avoidance of rheumatic fever, particularly of recurrent attacks, has been the subject of much discussion and many arguments for and against have been put forward by different authors. This operation had been performed on some of the patients in my material, and it is therefore perhaps of interest to investigate whether the prognosis was improved for tonsillectomized patients as compared with those not so treated. [Part of the present material has already been studied from this aspect by Landorf (1932).]

For this purpose I compiled tables 68 and 69, based on the total series of cases, exclusive of those cases where chorea was the primary manifestation since in the chorea group tonsillectomy was only performed in 4 cases after the initial attack (in 9 cases with chorea initially the operation had been done before the onset of the rheumatic infection). As will be seen from the tables, there seems to be no difference between cases where tonsillectomy had been performed before the onset of the initial attack and those tonsillectomized immediately after this attack (in a few cases the operation was not done immediately after the recovery from the initial attack), as compared with the remaining cases not submitted to tonsillectomy. The relation was the same whether one compared the recurrence rate with respect to no recurrences, one, two, or more recurrences, or the mortality rate. It should be mentioned, however, that the three deaths in the group in which tonsillectomy

Table 68.

Recurrence and mortality rates in all cases (not including those with chorea as the initial manifestation of rheumatic fever). Cases grouped according to time when tonsillectomy was performed.

Tonsillectomy performed at following stage:	No. of cases	No. of recurrences					Mortality
		0	1	2	3	≥4	
Before initial attack . .	33	20[2] 64.5 % ± 8.6	10[2] 32.2 % ± 8.4	—	—	1[2] 3.2 % ± 3.1	3 9.1 % ± 5.0
After initial attack[1] . .	66	37 56.1 % ± 6.1	20 30.3 % ± 5.7	5	4	—	8 12.1 % ± 4.0
				13.6 % ± 4.2			
Not performed[1]	387	211 54.6 % ± 2.6	120 31.0 % ± 2.4	34	11	11	38 9.8 % ± 1.6
				14.5 % ± 1.8			

was done before the initial attack include two patients who died at this attack. The lower mortality rate resulting from the deduction of these two cases nevertheless causes no statistically probable difference in the mortality between the three groups.

Considering now the severity of the cardiac affection at the follow-up (table 69) it will be found that from this aspect also, there are no differences whatsoever that cannot be ascribed to chance. Rather it must be said that despite the small number of cases in the two groups in which tonsillectomy was performed the agreement is nevertheless remarkably good.

It must, however, be admitted that presumably, the patients upon whom tonsillectomy was performed had tonsils in a particularly bad state and thus were especially susceptible to throat infections. If the tonsils had been allowed to remain in position this might possibly have been a factor favouring the appearance of recurrences, with the consequent increased risk of death or of severe cardiac disorder. The groups are therefore not absolutely

[1] Patients tonsillectomized after later attacks, as well as patients who died at initial attack, not included here.

[2] Percentage incidence calculated on the basis of 31 patients who survived the initial attack.

Table 69.

Cardiac affection in all cases (not including cases with chorea as the initial manifestation), which are grouped according to the time when tonsillectomy was performed.

Tonsillectomy performed at following stage:	Survivors examined at follow-up	Nature of cardiac affection			
		No heart disease / Possible heart disease	Functional classification		
			Class I	Class II	Class III
Before initial attack . .	30	7 + 6 = 13 43.3 % ± 9.1	15 50.0 % ± 9.1	2 6.7 % ± 4.9	—
After initial attack[1] . .	56	12 + 8 = 20 35.7 % ± 6.4	26 46.4 % ± 6.7	9 16.1 % ± 4.9	1 1.8 % ± 1.8
Not performed[1] . .	335	78 + 40 = 118 35.2 % ± 2.6	168 50.1 % ± 2.7	47 14.0 % ± 1.9	2 0.6 % ± 0.4

comparable. Only if every other patient had consistently been submitted to tonsillectomy would a material of an irreproachable scientific nature have been obtained.

Tonsillectomy was, of course, in some cases, performed after the appearance of a second or third attack of rheumatic fever. However, because of the small number of cases in this group (13 cases), as well as of the fact that relatively few of the patients in this material had more than one recurrence it is scarcely to be expected that a comparison between the various groups from this aspect would yield any information of value. Moreover, it seems unlikely that a tonsillectomy performed at a later stage than after the first attack would have a more favourable effect than an operation performed immediately after, or even before, the initial attack. No relevant information could, in fact, be obtained from a study of these cases.

[1] Patients tonsillectomized after later attacks, as well as patients who died at initial attack, not included here.

CHAPTER 9.

Is chorea minor in children a major manifestation of rheumatic fever?

In chapter 3 it was shown that a decline seems to have taken place in recent years in the number of patients who get chorea without previous evidence of rheumatic fever, while the same feature was not established with respect to patients who have arthritic symptoms or carditis alone as the initial manifestation of the disease. It was also found that the frequency of a complication such as pericarditis at some time during the subsequent course (at the initial attack or at a later recurrence) was very low (7.1 % $\pm$ 2.3 of 127 cases) in the chorea group, as compared with the frequency among both the patients with arthritic symptoms at the initial attack (19.8 % $\pm$ 1.8 of 521 cases) and above all those having only carditis initially (53.6 % $\pm$ 7.8 of 41 cases).

On the other hand, as regards the mortality (table 24), we find that the death rate is certainly low, at the initial attack, for the chorea group (0.79 per cent) as compared with a rate of 7.5 per cent for the group with arthritic symptoms at onset and 17.1 per cent for the group with carditis alone, but with respect to the mortality at later stages there is no essential difference in rate between the chorea group and the others. It therefore seems as though the course were milder, at the initial attack, in patients with chorea initially whereas, as regards the subsequent course, the death rate is approximately the same as in all other cases. (It should be stressed, however, that in the group with carditis alone, without any symptoms from the joints at the initial attack, the mortality at later attacks was higher than it was for the group with arthritic symptoms initially.)

Now, can any facts be adduced from the present investigation to support or refute the assumption that chorea minor in children is usually a true major manifestation of rheumatic fever? What differences or likenesses could be found in this investigation, between patients who had chorea as the initial manifestation and the other patients?

First as regards age and sex incidence, it was shown (p. 70) that when chorea constitutes the first evidence of rheumatic fever girls are in the majority over the boys, the proportion between them being as 7 to 3. The opposite was true of the cases where the initial attack took the form of arthritic symptoms, but the difference here was much less striking. In cases where neither arthritic nor choreiform symptoms could be established the proportion of girls was higher. On the other hand, if we consider all these cases as one unit, viz. rheumatic fever with various major manifestations, then the differences between boys and girls apparent in the subgroups are evened up, the slightly higher proportion of girls (51.2 per cent) as compared with boys (48.8 per cent) not being statistically significant.

In the chapter on recurrence, it was demonstrated that the recurrence rate with respect to the first recurrence was practically identical in the cases with chorea at the initial attack and in the cases with other manifestations (see fig. 13). On the other hand, a tendency was apparent in this material for the chorea cases to have their first recurrence after the lapse of a shorter period from the end of the initial attack than was the case among the other patients. After the passage of 8 years, however, this tendency had entirely disappeared and the same incidence as regards the first recurrence was then observed in both groups.

As was pointed out on page 114, the nature of the recurrence following on the respective initial manifestations varies to quite a considerable degree, although the most usual occurrence seems to be for the recurrent attack to take the same form as the initial manifestation. In all groups, however, there was a strong tendency for the patients to have a different type of manifestation at one of their later attacks, from that present at the initial attack (see table 28). Thus, in the group with chorea at the initial attack,

Table 70.

Showing all cases with recurrences in which the major manifestation at the initial attack and at each subsequent attack was chorea minor (with or without concurrent carditis).

Groups with following no. of recurrences	No. of cases	No. of cases examined at follow-up	No heart disease	Possible heart disease	Functional classification: Class I	Class II	Class III	Roentgenographic heart changes	Electrocardiographic changes
1	26[1]	23	5	0	14	4	—	7	5
			5		60.9 % ± 10.3	17.4 % ± 7.9		30.4 % ± 9.6	21.7 % ± 8.6
			21.7 % ± 8.6						
≥ 2	10[2]	9	1	1	5	2	—	4	3
			2		55.5 % ± 16.5	22.2 % ± 13.9		44.4 % ± 16.5	33.3 % ± 15.8
			22.2 % ± 13.9						
All cases . . .	36	32	6	1	19	6	—	11	8
			7		59.4 % ± 8.7	18.7 % ± 6.9		34.4 % ± 8.4	25.0 % ± 7.6
			21.9 % ± 7.3						

no less than 43.5 per cent of the cases having recurrences had an attack in the form of arthritic symptoms or carditis alone. In the group with arthritic symptoms initially, however, an attack of chorea occurred among the cases with recurrence in only 16.8 per cent, and similar conditions were found among the cases with carditis alone at the initial attack.

It is not uncommon, either, for a person with rheumatic fever to have arthritic symptoms or carditis alone initially, but later in the same attack to show signs of chorea; the arthritic symptoms, however, have usually disappeared before chorea makes its appearance. The opposite occurrence, namely that arthritic symptoms appear directly after an initial manifestation of chorea, is also ob-

[1] 1 of these cases not examined at follow-up; 2 deaths due to other causes than rheumatic fever.

[2] Of these, 5 cases had 2 recurrent attacks and 5 cases 4 or more; 1 case died 17 years after initial attack from vitium cordis with pulmonary infarction.

served. As may be seen from table 28, in my material these features were observed in connection with the initial attack, in no fewer than 29 cases (4.2 per cent). Similar features are to be observed in connection with recurrent attacks. (It should be pointed out, however, that in a few of these cases it is not absolutely clear whether the arthritic or the choreic symptoms constituted the primary manifestation.)

Finally, if we study the nature of the cardiac affection developing after the lapse of a long period (on an average, 12.2 years) from the onset of the initial attack (tables 43, 44, and 45), practically no differences whatsoever, as was shown on page 139, can be distinguished between the groups, either with respect to the nature of the cardiac affection, the functional capacity of the heart, or the roentgenographic and electrocardiographic heart changes. The same agreement is apparent when the two groups are compared from the standpoint of the occurrence or non-occurrence of recurrent attacks (table 48).

Table 70 shows those patients who at the initial attack and at each subsequent recurrence always had symptoms of chorea (with or without carditis), never arthritic symptoms or carditis alone. A comparison between this table and table 48 reveals that from this aspect also, there is no demonstrable difference between those cases where the major manifestation at recurrent attacks was chorea minor and those who had recurrences among the cases with arthritic symptoms or carditis alone at the initial attack.

The facts thus established seem to me to be undeniable evidence in favour of the view that chorea minor in childhood really may be a true major manifestation of rheumatic fever, although this form of the infection follows a much less severe course during the initial attack than the other major manifestations. To include these cases with chorea among the other rheumatic fever cases, as has been done in this study, would therefore seem to be fully justified.

CHAPTER 10.

Final conclusions.

The most important results obtained through this investigation may be briefly stated as follows:

1. Rheumatic fever usually appears for the first time in childhood and adolescence, 50 per cent of patients having their initial attack before the age of 20.

2. In childhood, the disease seems to attack both males and females indiscriminately, while at the older ages it shows a predilection for females.

3. Chorea minor does not as a rule occur as an initial manifestation after the age of 20. This major manifestation occurs predominantly in females. It seems to be a form of rheumatic fever that is strongly on the decline.

4. The mortality due to rheumatic fever is declining as the age at onset increases; in my material it was on the whole twice as high for the first 5 years of childhood as it was for the last 5 years (ending at 16 years). The incidence of pericarditis is also declining in proportion to increasing age at the onset of rheumatic fever. The rate for the first recurrence likewise decreases with increasing age at onset, this feature being particularly noticeable from the age of 13 years and onwards.

5. The death rate at the initial attack was 7.7 per cent on an average, but it was much higher (31.7 per cent) if carditis alone was present initially, and very low (0.79 per cent) if chorea constituted the primary lesion. Of the patients surviving the initial attack 10.2 per cent died at a later phase of the disease (the mean observation time for all cases was 12.2 years).

6. There has been a strong, gradual decline in the mortality, both as regards deaths at the initial attack and the death rate

for periods within 4 and 7 years of onset, in patients who had their first attack between 1922 and 1940. This decline was found to be statistically significant.

7. If the material be divided into two approximately equal halves, according to the year of the initial attack (i. e. the year-periods 1922—1931 and 1932—1940), the second half thus comprising mainly patients in whom the treatment time was prolonged until the erythrocyte microsedimentation rate had dropped to normal, the mortality in the 1922—1931 year-group shows a gradual rise in proportion to the appearance of recurrent attacks, while in the 1932—1940 year-group the death rate is approximately the same (or perhaps even a little lower) after the second attack as it is after the first; even on the appearance of further recurrences it shows only a moderate increase in the second year-group (see table 26).

8. Using the same system of classification, the recurrence rate is also lower for the 1932—1940 year-group than for the 1922—1931 year-group, both with regard to the first recurrence and, even more markedly, to the second and third recurrences. This decline in the recurrence rate on comparison of the two year-groups proved to be statistically probable.

9. The recurrence rate, with respect to the first recurrence, is highest during the first year (and particularly during the first half-year) after recovery from the initial attack, being up to the end of that period 14.4 % $\pm$ 1.5; at 10 years after the initial attack it is down to 1—2 per cent. The total recurrence rate (first recurrence) for the first 15 years is 48.6 per cent.

10. With respect to the risk of recurrence, no differences were distinguishable in patients with chorea (with or without carditis), arthritic symptoms (with or without carditis), or carditis alone at the initial attack; nor were there any differences with respect to the nature and degree of the cardiac disorder found at the follow-up examination.

11. Of the total number of survivors, 66.4 per cent had undoubted signs of cardiac disorder when examined at the follow-up on an average 12.2 years after the initial attack, 35.5 per cent of the total survivors had roentgenographically demonstrable

heart changes (about 25 per cent had roentgenologic enlargement of the heart), and 20.5 per cent had electrocardiographic alterations.

12. Corresponding to the decline in the mortality in the 1932—1940 year-group, as compared to the 1922—1931 year-group, there is a rise in the percentage of cases with cardiac disorder in the 1932—1940 year-group; fortunately, however, this increase applies to cases with slight cardiac disorder without limitation of physical activity (Class I), not to cases with the severer heart lesions.

13. The risk of permanent heart disease developing is less for patients having no recurrence than for those who have recurrences, while on the other hand this risk, as well as the risk of a serious heart lesion developing, rises in proportion to the number of recurrences.

14. The severity of the initial cardiac lesion is of significance insomuch as the mortality rate at later attacks increased in proportion to increasing severity of the initial carditis. The rate for the first recurrence and the incidence of permanent cardiac disorder or of severe heart lesions also rose in proportion to the severity of the initial cardiac affection.

15. In those cases complicated by pericarditis the mortality rate was 62 per cent in this material. Of all cases developing pericarditis, 84 per cent died within five years of the onset of the infection.

16. The development of an anemia (i. e. a hemoglobin value of less than 70 per cent according to the Autenrieth method) during the first three weeks of an initial attack of rheumatic fever is a bad omen, seeing that there was a statistically significant rise in the mortality in proportion to a decreasing hemoglobin content. Similarly, the rate for the first recurrence also rose proportionally to decreasing hemoglobin values. These observations do not, however, apply to patients whose initial attack was in the form of chorea, since the hemoglobin in these cases always registered normal values during the opening episode.

17. The appearance of subcutaneous nodules or erythema annulare is a bad sign, the mortality as well as the incidence of

cardiac disorder and severe cardiac lesions being higher when these symptoms are present at some stage of the disease. The incidence of pericarditis was much higher among the patients with nodules than it was among the other cases.

18. Tonsillectomy appears to be of no prognostic significance whatsoever, either when it is considered in relation to recurrence, mortality after recurrent attacks, or a future cardiac affection.

19. A closer analysis of the patients with chorea minor as the initial manifestation revealed that these cases did not diverge in any essential particulars from the others. Chorea minor in children thus appears to be a true major manifestation of rheumatic fever.

20. The reason for the, in many respects, improved prognosis is not absolutely clear. Thus, it has been shown that, in comparison with the custom in earlier years, there has been a definite, and statistically probable, tendency in recent years to bring the children to the hospital at an earlier phase of the disease, in consequence of which better possibilities present themselves for carrying out consistent rest in bed from the inception of the disease. Also, since the introduction of the policy of using the erythrocyte microsedimentation rate as a gauge of the presence or absence of an active infection the children have been kept in bed longer than previously, this observation applying chiefly to patients who had their initial attack of rheumatic fever during the second half of the 1922—1940 year-period. In addition, we have the fact that the after-care and follow-up controls have become more thorough and are extended over a longer period than was previously the custom. Although these circumstances may, to a greater or lesser extent, have contributed towards improving the prognosis, the possibility of an alteration in the *genus epidemicus* must nevertheless always be borne in mind. However, if there *is* such a continuous alteration in the *genus epidemicus*, then it is remarkable that it should not vary in accordance with the periodical variations in the incidence of the disease; because of this fact, the other factors advanced above may, perhaps, be attributed greater significance.

Summary.

The object of this investigation was to endeavour to obtain some idea of the prognosis, particularly the late prognosis, of rheumatic fever acquired in childhood, on the basis of a material composed of the majority of the patients from one particular geographical area in Sweden (the City of Gothenburg) and treated under conditions as closely approximating uniformity as could be expected. In this connection various questions relative to rheumatic fever have come up for study

After a brief description of the criteria used for diagnosis and a discussion on the same subject, the investigations of earlier authors are reviewed, particular reference being made to the aspect of the prognosis.

Chapter 1. The Material. The material used for this investigation consists of 689 cases of rheumatic fever, inclusive of chorea minor, treated at the Children's Hospital, Gothenburg, during the years 1922—1940. If the patients who have died are included, all cases with the exception of 18 (2.6 per cent) were available for a follow-up investigation (table 2).

At the follow-up investigation, which was carried out during the years 1944—1945, all the patients were submitted to a general examination, particular attention being paid to the condition of the heart. In all cases, therefore, roentgen examinations of the heart, and electrocardiographic examinations, were also made.

On the basis of the conditions at onset, the cases were divided into three groups, viz. cases with chorea[1], cases with arthritic symptoms[1], and cases with carditis alone at the initial attack (see tables 2, 27, and 28).

The length of the observation time for the cases submitted to the follow-up was at least four years in every instance. The average observation time was 12.2 years (table 3).

Chapter 2. Incidence of Rheumatic Fever from Various Viewpoints. *Age and sex incidence.* In this material approximately the same number of boys and girls had rheumatic fever, the girls being slightly

[1] With or without carditis. (Where chorea or arthritic symptoms are mentioned in the remainder of this summary the expression »with or without carditis» should always be understood.)

in the majority by a number statistically not probable. Taking into consideration only the cases with chorea as the initial manifestation of the disease, the number of girls was much above that of the boys, the proportion of boys to girls being as 3 to 7. On the other hand, when the initial attack took the form of arthritic symptoms, boys were in the majority. As regards carditis alone as the earliest evidence of the rheumatic infection, girls seemed to be in the majority.

The frequency curve for the various ages showed a consistent lag in incidence as regards the females, the peak incidence for males being at 9 years of age and that for females at 10 years of age. With respect to all cases (both children and adults) of rheumatic fever with the initial attack in the years 1922—1941, assembled from all the hospitals in Gothenburg, it was found that the percentage incidence of female cases (56.2 % ± 1.1) was considerably in advance of that of the males (43.8 % ± 1.1), the difference being statistically significant.

The frequency curve relative to the initial attack at various ages (fig. 1) showed a steady rise in the number of cases from the age of 1 up to and including the age of 9, after which age it declined again at approximately the same angle. After the age of childhood, the slope of the frequency curve became less steep.

As regards the age at which rheumatic fever is most likely to appear, it was found (table 6) that nearly two-fifths, or 39.5 per cent, of the patients had their initial attack before the age of 16, nearly one-half, or 49.5 per cent, before the age of 20, and nearly three-fourths, or 72.6 per cent, before the age of 30.

As regards cases with chorea minor as the initial manifestation of the disease, during the mentioned 20-year period, there was only one case under 4 years of age and only one over 20 years of age. The ages of predilection for chorea cases was found to be 7—12 years.

Annual and seasonal variations. The incidence of initial attacks of rheumatic fever showed a fairly considerable annual variation. The incidence of recurrent attacks also varied from year to year, the variations appearing, on the whole, to follow the variations in the incidence of initial attacks for the respective years. (Figs. 3, 4, and 5.)

Certain variations in the incidence of rheumatic fever were apparent for the different seasons, also. Thus, two minimum levels were observed for the periods February—April and July—September. The peak incidence occurred in the period November—January, but in addition to this there was a lower peak in May—June. As regards the incidence of recurrent attacks in the various months of the year, the relations were the same, though not so pronounced, as for the incidence of initial attacks.

The reasons for these variations, and in particular their possible connection with weather conditions and with other diseases such as scarlatina, are to be made the subject of discussion in a later publication.

Chapter 3. Has the Material changed Character in any way during the Course of Time? Can any Change in the Prognosis be detected for later Years? In the case of chorea as the primary manifestation of the rheumatic infection there was a definite falling tendency during the period 1922—1940, both with respect to the number of chorea cases and to the percentage incidence in relation to the total number of rheumatic fever cases, this decline being statistically significant. (There was no appreciable drop in the total number of children between the ages of 4 and 15 years in the City of Gothenburg for these years.)

No decline in the severity of the choreic symptoms, nor in the severity of the carditis present in some cases conjointly with the chorea, could be detected in this material.

As regards cases with other initial manifestations there was no decrease with respect to incidence, though on the whole, fairly pronounced fluctuations in incidence were to be observed. A certain falling tendency seemed, however, to be apparent in the percentage incidence of the severer cases, but this decline was mainly concentrated to the first part of the period covered by this investigation. There seemed also to be a certain drop in the number of cases sooner or later developing pericarditis, when considered according to the time of the initial attack, though this drop was not so great as to border on statistical probability.

On the other hand, as regards the mortality, both deaths at the initial attack and deaths within 4 and 7 years of onset, respectively, there was a strong decrease for the period 1922—1940, this decrease being statistically significant. This decline in the mortality was apparent both when the cases with chorea initially were included in the calculations and when they were excluded.

Reasons for this improvement in the prognosis are discussed, the necessity of bearing in mind the possibility of an alteration in the genus epidemicus being stressed. Another cause thought to play a contributory rôle is the gradually increasing tendency of parents to bring their children to the hospital at an early stage of the infection, which results in the children receiving the proper attention sooner than was previously the case. Still another reason may be that the children in the second half of the material were kept in bed for a longer period than the children in the first half; in other words, these patients were confined to bed until the erythrocyte microsedimentation rate had returned to normal.

Chapter 4. Mortality. *Causes of death.* The mortality due to the rheumatic infection or its aftermaths was 16.1 per cent (111 cases) in the series of cases discussed in this publication. In 3, possibly 4, cases blood culture revealed the presence of the *Streptococcus viridans* bacillus. In addition, we have a figure of 2.3 per cent for the rheumatic fever cases where death was due to some other cause.

Age at onset of infection and at death. As the age at onset increased the deaths tended to decrease. On the whole there were twice as many deaths when onset occurred in the first five years of childhood as when it occurred in the last five years, ending at 16 years.

Duration of disease in fatal cases. The mortality was highest for the year following the initial attack, approximately half the total number of deaths occurring before the end of the first year. For the second half-year and the three succeeding years, the mortality was much lower, remaining at about the same level for the whole of this period, after which it dropped rapidly.

When the material was divided up, according to the year of onset, into cases contracting the disease in the years 1922—1931 and those where the initial attack occurred in the years 1932—1940, it was found that the proportion of deaths was much lower in the later year-group, this feature being apparent both with regard to deaths within one year of onset and to deaths at a later stage (see fig. 12). The mortality for the first half-year subsequent to the initial attack was, in the second year-group (1932—1940), half the corresponding figure in the first year-group (1922—1931), while for the following four years the mortality in the second year-group had dropped to one-fourth of the corresponding rate in the 1922—1931 year-group.

Autopsy findings. A postmortem examination was carried out in 75 of the 127 patients who died. The findings from the heart are best described by referring to table 23.

Mortality in relation to type of initial manifestation and to number of attacks passed through. There was only one death at the initial attack among the 128 cases with chorea as the initial manifestation. Among the cases with arthritic symptoms at onset, the mortality at the initial attack was 7.5 per cent, while when carditis alone constituted the first evidence of the disease the mortality was no less than 31.7 per cent. The mortality from recurrent attacks, among cases surviving the initial attack, showed similar features, the only difference being that among the cases with chorea initially the mortality from recurrent attacks was approximately the same as that among the cases with arthritic symptoms initially.

The mortality for the whole series rose progressively in proportion to the number of attacks passed through. However, when the original material was divided up into two halves of approximately the same size this feature was found to apply only to the years 1922—1931, while for the years 1932—1940 no such rise was observable, except, perhaps, in connection with the third attack. As a contributory cause of this improvement in the prognosis for the later period (as regards death after recurrent attacks) the author suggests the longer period of treatment and the more adequate after-care that patients have begun to enjoy in more recent years.

CHAPTER 5. RECURRENCE. *Recurrence in relation to various initial manifestations.* Among the 671 patients in whom the course of the rheumatic infection was known, 47.2 per cent had one or more recurrences. The type of the recurrent attack was dependent in large measure on the nature of the initial manifestation. Among the patients who had chorea initially and then had recurrent attacks, no fewer than 77 per cent had chorea at at least one of the later attacks. As regards the cases with arthritic symptoms initially, two-thirds, or 68.4 per cent, had arthritic symptoms at at least one of the later attacks.

Of all the cases experiencing a recurrence, about 30 per cent had chorea at some attack, about 67 per cent had arthritic symptoms, and about 17 per cent had at least one attack with carditis alone.

Recurrence in relation to age at onset. When the cases were divided up into age-groups of three years the percentage incidence of patients having a recurrence was found to decline with increasing age of onset, the decline being fairly gradual in the lower age-groups while from the age of 13 years it was more rapid. While the percentage incidence of cases having one or more recurrences was 56.8 when onset occurred between the ages of 1 and 6 years, the corresponding figure for the ages 13—15 years was 29.2; in other words, in comparison with the lower age-group, only a little more than half as many patients in the higher age-group had one or more recurrent attacks.

Recurrence rate. A comparison between the cases with chorea minor initially and the other rheumatic fever cases showed that the recurrence rate in the two groups was almost identical, as far as the recurrence rate for a total period of 15 years from the initial attack was concerned. On the other hand, as regards the rate for a first recurrence within two years of the end of the initial attack, the figure seemed to be higher for the chorea cases. This difference disappeared entirely, however, for the years immediately following that period.

The recurrence rate judged on the basis of the whole material was highest during the first year following the end of the initial attack, 14.4 % $\pm$ 1.5, and then dropped gradually until, after a period of 10 years from onset, it was down to 1—2 per cent. The total recurrence rate was 34.1 per cent for the first 5 years after the end of the initial attack, 45.3 per cent for the first 10 years, and 48.6 per cent for the first 15 years. Thus, practically all first recurrences occurred within 10 years of the end of the initial attack.

A comparison of the recurrence rate in patients having the initial attack during the years 1922—1931 and in those in whom onset occurred in the years 1932—1940 revealed that the rate was lower for the latter group. This applied both to the rate for the first recurrence and, as far as could be judged, in an even greater degree to the second and third recurrence rates. The differences in the recurrence rates between the two year-groups were not sufficiently great, as regards each recurrence, to

be statistically significant, but when they were weighed together by a method suggested by Fisher the result must be said to be statistically probable. As a possible contributory cause of this decrease in the risk of recurrences in the later year-group it is suggested that the patients in this group (1932—1940) were kept in bed till the erythrocyte microsedimentation rate had returned to normal values while the patients in the earlier group (1922—1931) were only treated with bed rest till other clinical symptoms of active infection had disappeared. In addition to this, we have the fact that the after-care and follow-up control of patients have been more thorough and of longer duration in recent years.

An individual runs practically the same risk of having a fresh attack, irrespective of the number of attacks of rheumatic fever he has previously passed through.

Recurrence and mortality in relation to initial carditis. The severity of the initial cardiac affection is of great significance to the prognosis. Among the patients surviving their first attack, it was found that the more severe the cardiac affection at the initial attack the greater was the risk for a recurrence. The same features were apparent regarding the risk of death at a later attack. Among those patients who, at the initial attack, had cardiac involvement described as "severe," and who later had a recurrent attack (58.5 per cent), the mortality at this or a later attack was more than 50 per cent. On the other hand, of the patients without definite signs of cardiac affection at the initial attack, 34.4 per cent admittedly had a recurrence, but not one of these patients had died up to the time of the follow-up.

CHAPTER 6. PROGNOSIS AS REGARDS THE HEART. *Nature and degree of cardiac affection from a comparison between patients with initial attack during the periods 1922—1931 and 1932—1940, respectively.* The previously mentioned decline in the mortality in the 1932—1940 year-group as compared with the 1922—1931 year-group did not, as might be expected, imply an increase in the more severe heart lesions, nor, as may be seen from table 40, did it cause a progressive displacement backwards along the whole line resulting finally in an increase in the number of cases with no definitely established heart deformity. On the contrary, counterbalancing the decline in the proportion of deaths, an increase in the frequency of cases with established heart deformity was noted instead, the increase in the percentage incidence being about the same as the decrease in the mortality rate; this increase, however, applied almost solely to the group with the, from the functional aspect, mildest forms of cardiac affection (Class I, according to the Criteria Committee of the New York Heart Association).

Analagously to the heart lesions, the percentage incidence of roentgenographic heart changes was higher in the 1932—1940 year-group as compared with the figures for the 1922—1931 year-group, although here

the increase was not so striking as it was for the heart lesions. On the other hand, no such difference was observable with regard to the incidence of electrocardiographic changes.

Of the cases forming the whole material, one-fifth (22.2 per cent) were without signs of heart disease at the follow-up, while one-tenth (11.5 per cent) had "possible heart disease." In tables 41 and 42 are shown the incidence of various valvular lesions, as well as all patients divided up according to the functional capacity of the heart.

Nature and degree of cardiac affection and relation of cardiac affection to type of initial rheumatic manifestation. Regarding the cardiac affection found at the follow-up, no differences were apparent between cases with arthritic symptoms as the initial manifestation and those with chorea or carditis alone initially.

Cardiac affection at follow-up in relation to severity of initial carditis. Studying the cases without recurrence in my material I found that the more severe the cardiac affection at the initial attack the greater was the probability that a permanent heart lesion would result. The risk that this heart lesion would be of a severe nature also increased in proportion to the severity of the initial carditis. The presence of severe carditis at the initial attack does not necessarily mean, however, that a permanent heart lesion will develop, for even a severe carditis, if the rheumatic infection does not recur, can disappear entirely without leaving any traces. The milder the form of the initial carditis, however, the greater is the likelihood that the cardiac affection will disappear without deleterious effects.

It was interesting to note that of 21 chorea cases without recurrence, and without any definite signs of cardiac involvement during the active infection, 10 proved to have undeniable heart disorder at the follow-up.

Cardiac affection at follow-up in relation to age at onset. From this aspect there were no essential differences between the various age groups.

Frequency of heart disorder and its nature in relation to occurrence or non-occurrence of recurrent attacks. A comparison between the recurrence-free cases in the group with chorea initially and the other recurrence-free cases revealed no differences with respect to the heart affection at the follow-up. Practically 40 per cent of all recurrence-free cases had no signs of definite heart disease at the follow-up (24.5 per cent had no heart disease and 14.7 per cent were placed in the group of cases with "possible heart disease").

With the appearance of recurrences, the risk of sustaining permanent heart disease increased with each fresh recurrence passed through; the risk that a previous, mild heart lesion would become accentuated also increased. As the number of recurrences increased the number of cases without established heart lesions decreased, in fact, while there was a corresponding increase in the incidence of cardiac lesions causing

slight or marked limitation of physical activity (Classes II and III). Further, the incidence of roentgenographic enlargement of the heart, as well as of myocardial changes as evidenced by electrocardiograms, increased correspondingly in proportion to an increasing number of recurrences. All these deteriorations in the condition of the heart as the number of recurrences increased were found to be statistically significant.

It should be pointed out, however, that in some cases, even quite a number of recurrent attacks need not lead to permanent heart damage, at least not to judge from the course up to the time of the follow-up.

Chapter 7. Electrocardiographic Changes. Electrocardiographic alterations traceable to the rheumatic infection were encountered in this material in 109 cases out of 532 (20.5 per cent). Leaving out of account those cases where these changes were not unequivocally pathologic, 92 cases (17.3 per cent) remained.

Taking into consideration only the undeniable myocardial lesions observable on the electrocardiograms, 51.1 per cent of the above-mentioned 92 cases had an abnormal P-wave, 27.2 per cent showed prolongation of the P—Q interval, 32.6 per cent had an abnormal QRS complex, and 31.5 per cent an abnormal S—T segment or T-wave.

Roentgenographic changes in conjunction with electrocardiographic abnormality were present in 67 cases (61.5 per cent of the above 109 cases). Excluding those cases where the electrocardiographic tracings were not undeniably pathologic, 56 cases with both roentgenographic and electrocardiographic alterations remained, i. e. 60.8 per cent of the 92 cases with undoubted electrocardiographic abnormalities and 10.5 per cent of the total 532 cases submitted to the follow-up examination.

Chapter 8. Various other Signs and Aspects and their prognostic Significance. *Pericarditis.* The incidence of pericarditis in this material was 19.4 per cent (134 cases); in 17 cases, however, the condition was only discovered at autopsy. In cases where chorea constituted the initial manifestation of the rheumatic infection, however, the frequency of pericarditis in the subsequent course was only 7.1 per cent (only 3.9 per cent were detected clinically), while in cases where carditis alone was present at the initial attack a little over one-half (53.6 per cent) showed signs of pericarditis at some stage of the disease.

The tendency for pericarditis to develop in association with rheumatic fever was greatest when the disease first appeared in the first 5 to 6 years of life (25.6 per cent); for the following age-groups the incidence of this complication was around 20 per cent, while from the age of 13 it dropped noticeably to an average figure of 11 per cent.

A little over two-thirds (69.4 per cent) of all the patients with pericarditis had this complication in connection with the initial attack, while in the vast majority, 92.4 per cent, it had developed before the end of the second attack of rheumatic fever.

The previously noted improvement in the prognosis for patients who had their initial attack in the years 1932—1940, as compared with those contracting the disease between 1922 and 1931, applied to the pericarditis patients also, the mortality rate within 4 years of the initial attack showing a statistically probable decrease for the pericarditis group. On the other hand, the number of patients with signs of pericarditis at some time during their illness was practically identical in the two groups. This observed decline in the mortality was counterbalanced by an increase in the number of unequivocal heart lesions in the group without limitation of physical activity (Class I) a feature which also tallied with the relations observed in the whole material.

The risk of developing pericarditis in connection with rheumatic fever was found to be greatest at the first attack, and after that it declined as the number of attacks increased. The recurrence rate for these pericarditis cases seemed to be the same as for the whole material.

The mortality among patients developing pericarditis in this material was 61.9 % $\pm$ 4.2, this figure appearing to be approximately the same whether the complication arose at the initial attack or at a later attack. In 77.0 per cent of all the pericarditis patients who died, death occurred at the attack at which the pericarditis appeared.

Of the pericarditis patients who died, 56.6 per cent died within one year of the onset of infection and 84.3 per cent within 5 years.

In the pericarditis patients, the condition of the heart found at the follow-up examination was on the whole the same as that found for the whole material, although the number of cases without sure signs of permanent heart damage was much smaller in the pericarditis group, the difference being statistically significant.

There was nothing to indicate the presence of pericardial adhesion in these patients. No special investigations were, however, made to determine whether any such condition was present or not, but from the examinations made, there were no clinical, roentgenographic, or electrocardiographic findings that caused it to be suspected.

Pleural affection. Signs of pleurisy or pleural effusion were present in 4.5 per cent of the cases in the whole material. The commonest location was the left side; bilateral location came second, and a right-sided location was least common. There were no indications to suggest that tuberculous pleurisy might have been in question.

Subcutaneous nodules and erythema annulare. Subcutaneous nodules occurred at some time during the course of the disease in 5.4 per cent of the cases in this material, and erythema annulare in 5.8 per cent.

The incidence of pericarditis was approximately the same among the cases with erythema annulare as it was in the whole material, while among the cases with subcutaneous nodules this complication occurred much more frequently than it did in the whole series of cases (the difference was statistically probable).

The mortality was much higher when one or other of these symptoms was present, as compared with the mortality for the whole material.

The occurrence of cardiac disorder was higher among these cases than it was in the whole material, and in particular it was the severer heart lesions that increased.

On the other hand, the recurrence rate was approximately the same for these cases as for the whole material.

Microsedimentation rate of the red blood corpuscles. The erythrocyte microsedimentation rate was found to be very high in all cases unless the disease took the form of chorea, when it was usually less than 10 mm per hour. Neither from the maximum level of the reaction nor from the time elapsing between the onset of the infection and the return of the rate to less than 10 mm per hour could any information be obtained as to the prognosis with regard to the risk of recurrence or the probability of the development of permanent cardiac disorder. (For further details on this point, however, see pp. 182—185.)

Anemia. Among the 280 cases of rheumatic fever, exclusive of the chorea cases, that were followed by means of blood analysis 37.4 per cent (105 cases) showed signs of anemia (in this connection, a hemoglobin value of less than 70 per cent) at some time during their initial attack. In 5.4 per cent (of 280 cases) this finding was obtained for the first time after the lapse of more than three weeks from the initial attack.

The mortality among these 280 cases showed a statistically significant rise with decreasing hemoglobin. The frequency of pericarditis also increased with decreasing hemoglobin (actually, however, it is the anemia that develops, as a rule, in connection with a complicating pericarditis), and similarly, among these pericarditis cases, the mortality increased with a decreasing hemoglobin content. When pericarditis develops during the course of a rheumatic infection the prognosis as regards the mortality is much more hopeful if the hemoglobin registers normal values before, and during, this complication.

The recurrence rate also increased with decreasing hemoglobin values. On the other hand, the prognosis as to a future cardiac affection did not seem to be relatable to the presence or absence of an anemia.

As regards the 41 rheumatic fever cases where chorea was the initial manifestation, and where blood analyses were made, the hemoglobin registered more than 70 per cent during the whole initial attack. None of these cases subsequently died.

Tonsillectomy as a therapeutic measure in rheumatic fever. From the prognostic standpoint, the performance of tonsillectomy seemed to be of no significance, either when it was done before the onset of the infection or after the initial attack. The fact that, in most instances, it was in all probability the patients whose tonsils were more or less in a bad state who were submitted to tonsillectomy necessitates a certain amount of caution in the judging of these results, however.

CHAPTER 9. IS CHOREA MINOR IN CHILDREN A MAJOR MANIFESTATION OF RHEUMATIC FEVER? A comparison between those patients in whom the initial manifestation took the form of chorea minor and those in whom it took the form of arthritic symptoms with or without carditis, or of carditis alone, indicated that the former group was in most respects very similar to the latter group. The mortality at the initial attack was, however, very low for the chorea group in comparison with the other group while on the other hand the mortality at later attacks was approximately the same for the chorea cases as for the others. The incidence of pericarditis was also lower in the chorea group. On the other hand, the recurrence rate was practically the same in both groups. As regards the nature and severity of the cardiac affection observed at the follow-up, there were no differences at all between the two groups, either on comparison between recurrence-free cases and cases with one or more recurrences, or between those cases with only choreiform symptoms with or without concurrent carditis at each recurrent attack and those having recurrences in the group with other forms of initial manifestation. All these observations would seem to point strongly to the fact that chorea minor occurring in childhood is really a form of rheumatic fever.

CHAPTER 10. FINAL CONCLUSIONS. This chapter gives a general survey of the main results obtained in this investigation.

Bibliography.

Armstrong, D. B., and Wheatley, G. M.: Studies in rheumatic fever. Metrolife Insurance Company. New York 1944.

Ash, R.: Prognosis of rheumatic infection in childhood. Am. J. Dis. Child. *52:* 280, 1936.

Aschoff, L.: Zur Myocarditisfrage. Verhandl. d. deutsch. path. Gesellsch. *8:* 46, 1904.

Axén, O., Lindgren, E., and Malmström, G.: Contribution to the determination of measurement errors in the Liljestrand—Lysholm—Nylin—Zachrisson method of heart volume determination. Nord. Med. *29:* 592, 1946.

Bach, F., Hill, G. N., Preston, T. W., and Thornton, Ch. E.: Juvenile rheumatism in London. Ann. of Rheum. Dis. *1:* 210, 1939.

Ballonius, G. (Guillaume de Baillou): De reumatismo et pleuritide dorsale. In: Opera Omnia Medica, *4:* 275. Venetiis, Jacobi Thevart, 1736. (Cited by Wilson, 1940.)

Barlow, T., and Warner, F.: On subcutaneous nodules connected with fibrous structures occurring in children the subjects of rheumatism and chorea. Trans. Internat. Congress of Medicine. *4:* 116. London 1881.

Begbie, J.: Med. Chir. Soc. Edin., Febr. 17, 1847. (Republished in Contributions to Practical Medicine. Edin., 1862, p. 68.)

Berglund, H.: Hjärt-, kärl- och njursjukdomarnas behandling. Stockholm 1946.

Bergstrand, H.: Lärobok i patologi. Stockholm 1943.

Bindschedler, M. J. J.: Erythème annulaire rhumatismal de Lehndorff et Leiner chez un garçonnet. Bull. Soc. franç. de dermat. et syph., May 1936.

Bland, E. F., and Jones, T. D.: The delayed appearance of heart disease after rheumatic fever. J.A.M.A. *113:* 1380, 1939.

Blume, N. R.: Om sänkningsreaktionens förhållande vid några infektioner i barnaåldern. Svenska läk.-tidning No. 37, 1938.

Bonnier, G., and Tedin, O.: Biologisk variationsanalys. Stockholm 1940.

Bouillaud, Jean Baptiste: Traité clinique du rhumatisme articulaire et de la loi de coïncidence des inflammations du coeur avec cette maladie. Paris, J. B. Baillière, 1840.

Bouteille, E. M.: Traité de la chorée ou danse de St. Guy. Paris, J. B. Baillière, 1810.

BRIGHT, R.: Reports of medical cases selected with a view of illustrating the symptoms and cure of diseases by a reference to morbid anatomy, vol. 2, London, Longman, Rees, Orme, Brown & Green, 1831. (Cited by WILSON, 1940.)

BÄUMLER, CH.: Die deutsche Klinik am Eingange des 20. Jahrhunderts. Herausgegeb. v. LEYDEN u. KLEMPERER: Der akute Gelenkrheumatismus, vol. 2, 1903.

CABOT, R. C.: Facts on the Heart. Philadelphia 1926.

CAMPBELL, M., and WARNER, E. C.: A study of rheumatic disease in children. Lancet I, 1930, p. 61.

CHEADLE, W. B.: Albutt's System of Medicine, Macmillan and Co, London, 1897, III.

—— The various manifestations of the rheumatic state as exemplified in childhood and early life. Lectures delivered before the Harveian Society of London. London, Smith Elder & Comp., 1889. (Cited by WILSON, 1940.)

CHEINBERG, D. E.: Der Kampf gegen die rheumatische Infektion bei den Kindern in Sverdlovsk. La Pediatr. No. 5, pp. 95 and 152, 1937.

CHIARI, H.: Die pathologische Anatomie des akuten Rheumatismus, Verlag von Th. Steinkopf, Dresden and Leipzig, 1938.

CHOMEL, A. F.: Essai sur le rhumatisme. Paris 1813. (Cited by WILSON, 1940.)

—— Rhumatisme et goutte. Leçons de clinique médicale faites à l'Hotel Dieu de Paris, vol. 2, 1837. Translated into German by G. KRUPP, 1839.

CLARKE, P. J. H.: The clinical and public health aspects of juvenile rheumatism in Dublin. Irish J. M. Sc., March, 1940.

COBURN, A. F.: Observations on the mechanism of rheumatic fever. Lancet II: 1025, 1936.

COBURN, A. F., and MOORE, L. V.: The independence of chorea and rheumatic activity. Am. J. M. Sc. *193:* 1, 1937.

COHN, A. E., and LINGG, CL.: The natural history of rheumatic cardiac disease: A statistical study, J.A.M.A. *121:* 1, 1943.

COHN, A. E., and SWIFT, H. F.: Electrocardiographic evidence of myocardial involvment in rheumatic fever. J. Exper. Med. *39:* 1, 1924.

COOMBS, C. F.: Histology of rheumatic endocarditis. Lancet, I: 1377, 1909. (Cited by WILSON, 1940.)

—— Rheumatic heart disease. New York 1924.

DAHLBERG, G.: Statistical methods for medical and biological students. George Allen & Unwin Ltd, London 1940.

DAWSON, M. H.: Comparative study of subcutaneous nodules in rheumatic fever and rheumatoid arthritis. J. Exper. Med. *57:* 845, 1933.

DEBRÉ, R., LAMY, M., and JAMMET, M. L.: Érythème annulaire rhumatismal et streptococcémie à "Streptococcus viridans". Bull. et mém. Soc. méd. d. hôp. de Paris No. 26, Oct. 1936.

DEBRÉ, R.: Érythème annulaire et streptococcémie à "Streptococcus viridans" au cours de la maladie de Bouillaud. Arch. de méd. d. enf. *40:* 357, 1937.

Du Bois, E. F.: Basal metabolism in health and disease. London 1927.

Edén, L.: Transitory bradycardia during acute rheumatic carditis in children. Ann. Paed. *154:* 48, 1939.

Edström, G.: Febris rheumatica. Lund 1935.

Elghammar, H. W.: Erythrocyte sedimentation rate in rheumatic infection. Arch. Pediat. *51:* 281, 1934.

Fanconi, G., and Wissler, H.: Der Rheumatismus im Kindesalter. Part I. Der Rheumatismus Verus und Seine Differentialdiagnose, Verlag von Theodor Steinkopff, Dresden and Leipzig 1938.

Faxén, N.: La chorée et le rhumatisme. Rev. franç. de pédiat. *9:* 809, 1933.

Findlay, L.: The rheumatic infection in childhood, Edward Arnold & Co. London, 1931.

Fisher, R. A.: Statistical methods for research workers, 8th ed. London, 1941.

Friedlaender, A.: Acute rheumatic infection in children. Acta pædiat. XXXII: 393, 1945.

Friedlaender, A., and Mannheimer, E.: Clinical dates on rheumatic infection in childhood with special regard to the early diagnosis and to the value of the cardiac signs. Nord. Med. *24:* 2218, 1944.

Frühwald, R.: Dermatologie und Rheumatismus. Verlag von Theodor Steinkopff, Dresden and Steinkopff 1938.

Gerstley, J. R., Wile, S. A., Falstein, E. I., and Gayle, M.: Chorea: is it a manifestation of rheumatic fever? J. Pediat. *6:* 42, 1935.

Gezelius, G.: Die Anaemie bei akutem rheumatischen Fieber im Kindesalter. Acta pædiat. *23:* 361, 1939.

Gibson, S., and Denenholz, E. J.: Rheumatic heart disease. J. Pediat. *9:* 505, 1936.

Giroux, Léon.: Les Rhumatismes aigus et leur traitement. J. B. Baillière & Fils. Paris 1923.

Glanzmann, E.: Die rheumatische Infektion im Kindesalter. Verlag von Georg Thieme, Leipzig 1935.

Grenet, H.: La maladie rhumatismale chez l'enfant. Arch. de méd. d. enf. *40:* 329, 1937.

—— Notes cliniques sur la maladie rhumatismale (maladie de Bouillaud) chez l'enfant. Bull. Soc. méd. d. hôp. de Paris, III, pp. 53 and 701, 1937.

Grenet, H., and Joly, F.: Les formes graves de rhumatisme cardiaque dans l'enfance. Bull. méd. Paris, p. 305, 1938.

Hawkins, F. B.: Rheumatism and some diseases of the heart and other organs (Gulstonian lectures). London 1826. (Cited by Hedley, 1940.)

Hedley, O. F.: Rheumatic heart disease in Philadelphia hospitals: II. Pub. Health Rep. *55:* 1647, 1940.

—— Salient public health features of rheumatic heart disease. Pub. Health Rep. *52:* 164, 1937.

Heffer, E. T.: The electrocardiogram in rheumatic fever. A study of Sydenham's chorea. J. Pediat. *18:* 363, 1941.

HELMREICH, E.: Der Rheumatismus im Kindesalter und seine Behandlung, Ferd. Enke Verlag, Stuttgart 1935.

HILLIER, T.: Diseases of children. J. Walton, London, 1868.

HOLT, L. E.: Diseases of infancy and childhood. New York, p. 1102, 1922.

JOHN, J., and NOBEL, E.: Über die Prognose der rheumatischen Vitien im Kindesalter. Zeitschr. f. Kinderh. *36:* 335, 1923.

JONES, T. D.: Heart disease in childhood. Am. J. Pub. Health *28:* 637, 1938.

—— The diagnosis of rheumatic fever. J.A.M.A. *126:* 481, 1944.

JONES, T. D., and BLAND, E. F.: Clinical significance of chorea as a manifestation of rheumatic fever. J.A.M.A. *105:* 571, 1935.

JONES, T. D., and MOTE, J. R.: The clinical importance of infection of the respiratory tract in rheumatic fever. J.A.M.A. *113:* 898, 1939.

JONSELL, S.: A method for determination of the heart size by teleroentgenography (a heart volume index). Acta radiol. *20:* 325, 1939.

JUCA, A., and WHITE, P. D.: The cause of death in rheumatic heart disease in adults. J.A.M.A. *125:* 767, 1944.

KAHLMETER, G.: Le sang dans le rhumatisme chronique progressiv généralisé. Conférence Scientifique Internationale Du Rhumatisme Chronique Progressiv Généralisé. Aix les Bains, 1934.

—— The sedimentation rate of erythrocytes in various rheumatic conditions. Medical Journ. and Record, Nov. 7, 1928.

KAISER, A. D.: Factors that influence rheumatic disease in children. Based on a study of 1,200 rheumatic children. J.A.M.A. *103:* 886, 1934.

KARLSTRÖM, F.: Frekvensen av chorea minor och polyarthritis acuta hos barn under de sista 20 åren i Stockholm samt några av sjukdomarnas kliniska drag. Nord. Med. *8:* 2653, 1940.

KLINGE, F.: Der Rheumatismus in Lubarsch O. und von Ostertag R.: Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie des Menschen und der Tiere. Verlag von J. F. Bergmann. München, *27*, 1933.

KRAFFT-EBING: Der Akute Gelenkrheumatismus. (Cited by ROLLY, 1920.)

LANDAU, A.: Microsedimentation (Linzenmeier—Raunert method). Am. J. Dis. Child. *45:* 691, 1933.

LANDORF, N.: Akute Polyarthritis und Tonsillektomie. Acta Ped. *XII:* 212, 1932.

LEBERT: Klinik des akuten Gelenkrheumatismus. Erlangen 1860. (Cited by ROLLY, 1920.)

LEHNDORFF, H., and LEINER, C.: Erythema annulare, Ein typisches Exanthem bei Endocarditis. Ztschr. f. Kinderh. *32:* 46, 1922.

LEICHTENTRITT, B.: Die rheumatische Infektion im Kindesalter. Ergebn. d. inn. Med. u. Kinderh. *37:* 1, 1930.

—— Der akute Gelenkrheumatismus als Teilerscheinung der rheumatischen Infektion, zugleich ein Beitrag zum Rheumatismus nodosus und zur Stillschen Krankheit. Pfaundler & Schlossman. Handb. der Kinderh., Vol. II, Ed. 4, 1931.

LILJESTRAND, G., LYSHOLM, E., NYLIN, G., and ZACHRISSON, C. G.: Normal heart volume in man. Am. Heart J. *17:* 406, 1939.

LILJESTRAND, G., LYSHOLM, E., and NYLIN, G.: The immediate effects of muscular work on the stroke and heart volume in man. Scandinav. Arch. f. Physiol. *80,* 265, 1938.

MANNHEIMER, E.: Calibrated phonocardiography and electrocardiography. Acta Pæd. XXVIII, suppl. II.

MARTIN, A. T.: Twenty years' observation of 1,438 children with rheumatic heart disease. J.A.M.A. *117:* 1663, 1941.

MERRITT, K.: Rheumatic nodules. Their incidence in rheumatic infections. Am. J. Dis. Child. *35:* 823, 1928.

NATTENHEIMER, R.: Das Verhalten des Erythema Annulare und der Noduli Rheumatici im Verlaufe des Rheumatismus Verus. Inaugural-Diss., Zürich 1939.

NATHHORST, H.: Nierenreizung bei rheumatischen Fieber im Kindesalter. Ann. Paed. *155:* 285, 1940.

NOBÉCOURT, P.: Le salicylate de sodium dans la maladie de Bouillaud chez les enfants. Phare Médical de Paris 1935.

Nomenclature and criteria for diagnosis of diseases of the heart. By the Criteria Committee of the New York Heart Association. New York 1939 and 1945.

NORDGREN, R.: Sur le mode d'apparition de la chorée. Acta pæd. II: 164, 1922.

ORGAIN, E. S., and MARTIN, J. M.: Electrocardiographic alterations by rheumatic fever. Am. J. Dis. Child. *62:* 26, 1941.

OSLER, W.: Principles and practice of medicine. New York, 1920.

—— On chorea and choreiform affections. London, H. K. Lewis & Co. 1894.

PAYNE, W. W., and SCHLESINGER, B.: A study of the sedimentation rate in juvenile rheumatism. Arch. Dis. Childhood. *10:* 403, 1935.

PITCAIRN, D.: Cited by WILSON, 1940.

POYNTON, F. J., PATERSON, D., and SPENCE, J. C.: Acute rheumatism in children under twelve years of age. Lancet *199:* 1086, 1920.

PRIBRAM: Der akute Gelenkrheumatismus in Nothnagels spez. Pathologie und Therapie, Vol. V: 1, Vienna 1899.

REID, W. D., and KENWAY, F. L.: Value of electrocardiogram in acute rheumatic fever. New England J. Med. *198:* 177, 1928.

RICKER, G.: Das Zentralnervensystem und die Rheumatisch genannte akute Polyarthritis mit ihrem Zubehör, Verlag von Theodor Steinkopff, Dresden and Leipzig 1938.

RIESS: Polyarthritis rheumatica in Realenzyklopädie der gesamten Heilkunde. *19:* 258, 1898.

ROGER, H.: Observations et considérations sur le rhumatisme chez les enfants. Archives générales de Méd. I, vol. 9, p. 54. Paris 1867.

—— Observations et considérations sur le rhumatisme chez les enfants. Deuxième partie: Les maladies du coeur chez les enfants, et spécialement de leurs rapports avec la chorée (chorée cardiaque). Archives générales de Méd. I, vol. 10, p. 26. Paris 1868.

ROLLY, F.: Der akute Gelenkrheumatismus nebst Chorea minor und Rheumatoide. Verlag von Julius Springer, Berlin 1920.

ROTH, IRVING R., LINGG, CLAIRE, and WHITTEMORE, ALICE: Heart disease in children. A. Rheumatic group. I. Certain aspects of the age at onset and of recurrences in 488 cases of juvenile rheumatism ushered in by major clinical manifestations. Am. Heart J. *13:* 36, 1937.

SCHERF, D., and BOYD, L. J.: Clinical electrocardiography, W. Heinemann, London 1943 and 1945.

SCHLESINGER, B.: The Milroy Lectures 1938 on the public health aspect of heart disease in childhood. Lancet, pp. 593 and 649, 1938.

SÉE, GERMAIN: Mém. de l'acad. de Méd. Paris 1850, XV, 373. (Cited by FINDLAY, 1931.)

SENATOR: Die Krankheiten des Bewegungsapparates in v. Ziemssens Handb. der spez. Path. u. Ther. *13:* 1, 1875.

SHAPIRO, M. J.: Electrocardiographic changes in quiescent rheumatic disease in children and young adults. Am. Heart J. *5:* 504, 1930.

Standardization of praecordial leads. Joint recommendations of the American Heart Association and the Cardiac Society of Great Britain and Ireland. Am. Heart J. *15:* 107 and 235, 1938.

STRANSKY, E.: Hämatologie des Kindesalters, Baar-Stransky 1928, p. 347. (Cited by GEZELIUS 1939.)

STROUD, W. D., GOLDSMITH, M. A., POLK, D. S., and THORP, F. Q.: Ten years' observation of children with rheumatic heart disease. J.A.M.A. *101:* 502, 1933.

STROUD, W. D., and TWADDLE, P. H.: Fifteen years' observation of children with rheumatic heart disease. J.A.M.A. *114:* 629, 1940.

SUTTON, L. P.: Observations on certain etiological factors in rheumatism, Am. Heart J. *4:* 145, 1938.

SWIFT, H. F. in CECIL R. L. F.: Text-Book of Medicine, ed. 2, Philadelphia, W. B. Saunders Company 1931.

SWIFT, H. F.: Public health aspects of rheumatic heart disease. J.A.M.A. *115:* 1509, 1940.

—— Rheumatic fever. J.A.M.A. *92:* 2071, 1929.

SYDENHAM, T.: Works of Thomas Sydenham, M. D. Translated from the Latin edition of Dr. Greenhill, with a life of the author by R. G. LATHAM, M. D. London, printed for the Sydenham Society 1848.

VAUBEL, E.: Der akute Gelenkreumatismus. Verlag von Theodor Steinkopff, Dresden and Leipzig 1938.

VOELCKER, A. F.: Diseases of children, Garrod, Batten and Thursfield, London 1913.

WALLGREN, A.: Acute rheumatic fever in children. Irish J. Med. Sc. Nov. 1939.

—— Studies on erythema annulare rheumaticum. Acta pediat. XVII: 447, 1935.

—— Rheumatisk endocardit vid scarlatina. Svenska läk.-tidning No. 21, 1939.

WARBURG, E.: Drag av den reumatiska infektionens klinik. Ugesk. f. læger *93:* 1031 and 1051, 1931.

WATSON, T.: Observations on rheumatism of the heart. London, M. Gaz. 16: 56—64, 91, 164; 1835. (Cited by WILSON, 1940.)

WEDMAN, A. G., and WEDMAN, B. G.: Rheumatic infections in Cincinnati hospitals. Am. J. Dis. Child. *67:* 182, 1944.

WELLS, W. C.: On rheumatism of the heart. Trans. Soc. for Improvement Medical and Chirurgical Science. London 1812.

WILSON, MAY: Rheumatic fever, The Commonwealth Fund, New York 1940.

WILSON, M. G., and LUBSCHEZ, R.: Recurrence rates in rheumatic fever. J.A.M.A. *126:* 477, 1944.

WOLLENBERG: Infectiöse Chorea (Chorea minor) in Nothnagels spezielle Pathologie und Terapie, Vol. 12, H. 2, p. 9, 1895.

WYCKOFF, J., and LINGG, CLAIRE: Statistical studies bearing on problems in classification of heart disease, Am. Heart J. I: 446, 1926.

Contents.

PREFACE 5

INTRODUCTION 7

CRITERIA FOR DIAGNOSIS 10
- Comments on the major manifestations 11
- Comments on the minor manifestations 17

EARLIER INVESTIGATIONS 21
- Age and sex incidence 21
- Annual and seasonal variations 26
- Incidence of various major manifestations 29
- Prognosis 31
  - Mortality 32
  - Recurrence rate 36
  - Prognosis as regards the heart 40

THE AUTHOR'S INVESTIGATION 51

CHAPTER 1. *The material* 51
- Its composition and limitations 51
- The follow-up investigation 53
  - Its plan and effectiveness 53
  - Methods 57
- Classification 59
- Observation time 61
- Statistical methods 61

CHAPTER 2. *Incidence of rheumatic fever from various viewpoints* 63
- Age and sex incidence 63
- Annual and seasonal variations 70

CHAPTER 3. *Has the material changed character in any way during the course of time? Can any change in the prognosis be detected for later years?* 77

CHAPTER 4. *Mortality* 97
- Causes of death 97
- Age at onset of infection and at death 100
- Duration of disease in fatal cases 103
- Autopsy findings 106
- Mortality rate in relation to type of initial manifestation and to number of attacks passed through 108

CHAPTER 5. *Recurrence* 113
Definition 113
Recurrence in relation to various initial manifestations 114
Recurrence in relation to age at onset 116
Recurrence rate 117
Methods 117
Results 119
Is there any change in the recurrence rate in this material? 123
Comparisons between different attacks in respect of the risk of further recurrence 131
Recurrence and mortality in relation to severity of initial carditis 132
CHAPTER 6. *Prognosis as regards the heart* 135
Nature and degree of cardiac affection from a comparison between patients with initial attack during the periods 1922—1931 and 1932—1940, respectively 136
Nature and degree of cardiac affection and relation of cardiac affection to type of initial rheumatic manifestation 139
Cardiac affection at follow-up in relation to severity of initial carditis 144
Cardiac affection at follow-up in relation to age at onset 147
Frequency of heart disorder and its nature in relation to occurrence or non-occurrence of recurrent attacks 148
CHAPTER 7. *Electrocardiographic changes* 154
CHAPTER 8. *Various other signs and aspects and their prognostic significance* 162
Pericarditis 162
Frequency 162
Age at onset 165
Is there any change in the prognosis for patients who contracted pericarditis similar to that described for the whole series? 167
Mortality and recurrence rate 169
Condition of heart at follow-up 173
Pleural affection 176
Subcutaneous nodules and erythema annulare 178
Microsedimentation rate of the red blood corpuscles 180
Anemia 187
Tonsillectomy as a therapeutic measure in rheumatic fever .. 192
CHAPTER 9. *Is chorea minor in children a major manifestation of rheumatic fever?* 195
CHAPTER 10. *Final conclusions* 199
SUMMARY 203
BIBLIOGRAPHY 214

MEDICAL LIBRARY
JUN 5 1947

ACTA PÆDIATRICA

EDITORES:

A. LICHTENSTEIN, STOCKHOLM. A. WALLGREN, STOCKHOLM

REDACTORES:

IN DANIA: BENT ANDERSEN, AARHUS, OLUF ANDERSEN, KØBENHAVN, C. E. BLOCH, KØBENHAVN, P. PLUM, KØBENHAVN. *IN FENNIA:* P. HEINIÖ, HELSINGFORS, V. RANTASALO, HELSINGFORS, C.-E. RÄIHÄ, HELSINGFORS, T. SALMI, ÅBO, ARVO YLPPÖ, HELSINGFORS. *IN HOLLANDIA:* E. GORTER, LEIDEN, CORNELIA DE LANGE, AMSTERDAM, J. VAN LOOKEREN CAMPAGNE, GRONINGEN. *IN NORVEGIA:* TH. FRÖLICH, OSLO, LEIF SALOMONSEN, OSLO, L. STOLTENBERG, OSLO, A. SUNDAL, OSLO, KIRSTEN UTHEIM-TOVERUD, OSLO. *IN SUECIA:* C. GYLLENSWÄRD, UPPSALA, N. MALMBERG, STOCKHOLM, STURE SIWE, LUND, WILHELM WERNSTEDT, STOCKHOLM, Y. ÅKERRÉN, GÖTEBORG.

REDIGENDA CURAVIT
A. LICHTENSTEIN
KRONPRINSESSAN LOVISAS BARNSJUKHUS,
STOCKHOLM

Vol. XXXIII. Supplementum VII
1946

Almqvist & Wiksells Boktryckeri Aktiebolag
UPPSALA 1946

ACTA PÆDIATRICA

PROFESSOR A. LICHTENSTEIN
KRONPRINSESSAN LOVISAS BARNSJUKHUS,
30 POLHEMSGATAN, STOCKHOLM

The 'ACTA PÆDIATRICA' contain articles relating to pediatrics. These articles are published in English, French or German, according to the wishes of the author. Each number consists of about 6 printed sheets, 4 numbers forming a volume. The numbers will be issued as soon as the articles sent in can be printed. The 'Acta' is open to articles from foreign authors in all countries, if sufficient space can be found for them. Manuscripts are to be sent direct to the Editor, to whom also enquiries about the exchanging of papers are to be directed. The subscription should be forwarded to the Editor. Each volume costs 25 Swedish crowns or 25 shillings or 5 dollars.

ACTA PÆDIATRICA enthalten Arbeiten aus dem Gebiete der Kinderheilkunde. Die Arbeiten werden, je nach eigener Wahl des Verfassers, in deutscher, französischer oder englischer Sprache veröffentlicht. Jedes Heft enthält cirka 6 Druckbogen; 4 Hefte bilden einen Band. Die Hefte erscheinen, je nachdem die in dieselben aufzunehmenden Aufsätze druckfertig vorliegen. Die Acta nehmen nach Möglichkeit auch Arbeiten ausländischer Verfasser aller Nationen auf. Manuskripte nimmt der Herausgeber entgegen, desgleichen Wünsche betreffs Austausch von Zeitschriften. Abonnementanmeldung bei dem Herausgeber. Preis pro Band 25 schwedische Kronen.

Les ACTA PÆDIATRICA contiennent des ouvrages du domaine de la pédiatrie. Les études sont publiées en français, anglais ou allemand au choix de l'auteur. Chaque fascicule contient env. 6 feuilles in-8°; 4 fascicules forment un volume. Les fascicules paraissent au fur et à mesure que les articles y destinés sont imprimés. Les Acta reproduisent, dans la mesure du possible, les articles d'auteurs étrangers de tous les pays. Les manuscrits doivent être expédiés à l'éditeur, à qui les demandes relativement à l'échange de journaux devront également être adressées. Abonnement chez l'éditeur. Prix par volume Cr. Suéd. 25.

ACTA PÆDIATRICA

Acta Chirurgica Scandinavica. *Editorial Board: in Denmark* P. N. Hansen, S. Kjærgaard; *in Finland* R. Faltin, F. Langenskiöld; *in Norway* J. Holst, J. Nicolaysen; *in Sweden* E. Key (Editor), G. Petrén; *in Iceland* G. Thoroddsen, Reykjavik. Subscription: 25 Sw. crowns. Address: Tryckerigatan 2, Stockholm.

Acta Dermato-Venereologica. *Editorial Board: in America* H. Goodman; *in Czecho-Slovakia* F. Šamberger; *in Denmark* C. Rasch; *in England* H. Mac Cormac; *in Holland* S. Mendes Da Costa; *in Norway* H. P. Lie; *in Sweden* J. Almkvist, Sven Hellerström (Editor); *in Switzerland* Ch. Du Bois. Subscription: 25 Sw. crowns. Address: Sturegatan 22, Stockholm.

Acta Medica Scandinavica. *Editorial Board: in Denmark* H. I. Bing, K. Faber, Eggert Möller, C. Sonne, Erik Warburg; *in Finland* Gösta Becker, R. Ehrström, Östen Holsti, F. Saltzman; *in the Netherlands* A. A. Hijmans van den Bergh, W. A. Kuenen, L. Polak Daniels, P. Ruitinga, I. Snapper; *in Norway* Olav Hanssen, S. B. Laache, H. A. Salvesen, Olav Scheel; *in Sweden* G. Bergmark, I. Holmgren (Editor), Sven Ingvar. Subscription: 20 Sw. crowns in the Scandinavian countries and 25 Sw. crowns in other countries. Address: Acta Medica Scand., Stockholm.

Acta Obstetricia et Gynecologica Scandinavica. *Editorial Board: in Denmark* E. Hauch; *in Finland* S. E. Wichmann; *in Norway* A. Sunde; *in Sweden* Erik Ahlström (Editor). Subscription: 25 Sw. crowns. Address: Stockholm 5.

Acta Oto-Laryngologica. *Editorial Board: in Denmark* E. Schmiegelow; *in Finland* Y. Meurman; *in Holland* H. Burger; *in Norway* F. Leegaard; *in Sweden* G. Holmgren (Editor), G. Öhngren; *in Switzerland* F. R. Nager; *in Hungary* Z. de Lénárt. Subscription: 25 Sw. crowns. Address: Hospital Sabbatsberg, Stockholm.

Acta Odontologica Scandinavica. *Editorial Board: in Denmark* E. Faber; *in Finland* J. Kivimaki; *in Norway* O. Grythe; *in Sweden* G. Y. Hildebrand (Editor). Subscription: 20 Sw. crowns. Address: Tryckerigatan 2, Stockholm.

Acta Pædiatrica. *Editorial Board: in Denmark* Bent Andersen, Oluf Andersen, C. E. Bloch, P. Plum; *in Finland* P. Heinio, V. Rantasalo, C.-E. Räihä, T. Salmi, Arvo Ylppö; *in Holland* E. Gorter, Cornelia de Lange, J. van Lookeren Campagne; *in Norway* Th. Frölich, Leif Salomonsen, L. Stoltenberg, A. Sundal, Kirsten Utheim-Toverud; *in Sweden* C. Gyllenswärd, A. Lichtenstein (Editor), N. Malmberg, Sture Siwe, Arvid Wallgren, Wilh. Wernstedt, Y. Åkerrén. Subscription: 25 Sw. crowns. Address: Polhemsgatan 30, Stockholm.

Acta Radiologica. *Editorial Board: in Denmark* P. Flemming Møller, A. Reyn; *in Finland* C. G. Jansson, G. A. Wetterstrand; *in Holland* L. G. Heilbron, J. W. S. Henkensfeldt Jansen; *in Norway* S. A. Heyerdahl, H. Thue; *in Sweden* L. Edling; *in Switzerland* R. Gilbert, H. Schinz. Editor: G. Forssell: Sophiahemmets Röntgeninstitut, Stockholm. Subscription: 25 Sw. crowns. Address: Tryckerigatan 2, Stockholm.

Acta Ophthalmologica. *Redactores:* Fredrik Berg, Uppsala. Sven Larsson, Lund. Emil Enroth, Helsingfors. Birger Malling, Oslo. Ejler Holm, Kobenhavn. Hans Ulrik Möller, Kobenhavn. Ingolf Schiötz, Oslo. Mauno Vannas, Helsingfors. *Edenda curaverunt:* Ejler Holm, Hans Ulrik Möller. Subscription: Dan. Cr. 35.—.

Acta Orthopaedica Scandinavica. *Fundator:* Patrik Haglund. *Redactores:* P. G. K. Bentzon, Aarhus. O. Chiewitz, Kobenhavn. G. Frising, Lund. Poul Guildal, Kobenhavn. Sven Johansson, Göteborg. F. Langenskiöld, Helsingfors. E. Platou, Oslo. H. Sundt, Stavern, Norway. H. Waldenström, Stockholm. *Redigenda curavit:* P. G. K. Bentzon, Kathrinebjergvej 2, Aarhus, Denmark. Subscription: Dan. Cr. 35.—.

Acta Pathologica et Microbiologica Scandinavica. *Redactores:* C. G. Ahlström, Lund. H. Holth, Oslo. K. A. Jensen, Kobenhavn. A. Lindau, Lund. Poul Møller, Kobenhavn. Osv. Renkonen, Helsingfors. Georg Waaler, Oslo. Axel Wallgren, Helsingfors. *Redigenda curavit:* Tage Kemp, Tagensvej 14, Kobenhavn. Subscription: Dan. Cr. 35.—.

Acta Pharmacologica et Toxicologica. Iussu Societatis Pharmacologicae Hafniae Edita. *Redactores:* Gunnar Ahlgren, Lund. Erik Jacobsen, Kobenhavn. Armas Vartiainen, Helsinki. *Redigenda curavit:* Knud O. Møller, Kobenhavn. Subscription: Dan. Cr. 35.—.

Acta Psychiatrica et Neurologica. *Redactores:* Nils Antoni, Stockholm. B. Brouwer, Amsterdam. E. Essen-Möller, Lund. Harald Fabritius, Helsingfors. Mogens Fog, Kobenhavn. Hjalmar Helweg, Kobenhavn. Sv. Ingvar, Lund. G. H. Monrad-Krohn, Oslo. Matti Kaila, Helsingfors. H. Sjöbring, Lund. Cornelis Winkler, Utrecht. *Redigenda curavit:* Knud H. Krabbe, Kobenhavn, Dr. Tværgade 6. Subscription: Dan. Cr. 35.—.

Acta Tuberculosea Scandinavica. *Redactores:* S. Bang, Kobenhavn. Axel von Bonsdorff, Nummela (Finland). J. Heimbeck, Oslo. A. Kristenson, Stockholm. Sig. Magnússon, Reykjavik. H. Mollgaard, Kobenhavn. John Lundquist, Stockholm. Alex. Tuxen, Vardasen (Norway). *Editor:* Dr. med. Niels Sjörslev, St. Strands-træde 21, Kobenhavn K. Subscription: Dan. Cr. 35.—.

Subscriptions and advertisement for these Acta should be forwarded under the names of the respective Acta, address: Einar Munksgaard, Norregade 6, Copenhagen. Manuscripts to be forwarded to the Editor or the redigenda curavit.

EINAR MUNKSGAARD — COPENHAGEN

INDEX ACTORUM.

ERIK JACOBSSON: Rheumatic Fever with Chorea Minor. A clinical study with special reference to the prognosis . . . . . 1—222